PROPORTIONAL YIELDS AND PROCESSING OF PORK DERIVED FROM DIFFERENT HALOTHANE HYPERTHERMIA PIG GENOTYPES

by

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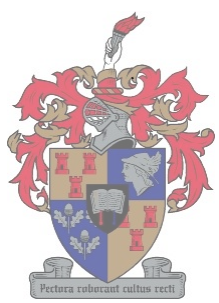

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DECLARATION

I, the undersigned, hereby declare that the work contained in this dessertation is my own original work and that I have not previously in its entirety or in part submitted it at any university for a degree.

SUMMARY

The carcass and meat quality characteristics of three halothane genotypes in pigs were evaluated. Sixty crossbred Landrace x Large White pigs (NN = 25, Nn = 19, nn = 16) of ± 86 kg live weight were slaughtered, the carcasses chilled for 24 h at 2°C, certain carcass and meat quality traits determined and the shoulder and leg cuts deboned and devided into primal cuts. Least squares (LS) means for dressing percentage of the Nn genotypes was higher ($P < 0.05$) than that of the NN and nn genotypes. LS means for carcass length was highest ($P < 0.05$) for carcasses derived from the nn genotype. The LS means calculated for several measurements on the split carcasses (midline fat measurements) and cross sections between the 2nd and 3rd last thoracic vertebrae relating to fat depositioning (45 mm from the midline) and lean depositioning (eye muscle width, depth and area) provided significant evidence that the nn genotype yielded leaner carcasses than Nn and NN genotypes. Fitting prediction equations to some of the these measurements to calculate LS means for predicted lean yield confirmed that nn genotypes yielded carcasses with less fat and more lean.

Further dissecting of selected cuts (shoulders and legs) also showed that the LS means for bone, fat and lean yield from nn genotypes were more desired than those from the other two genotypes, with probabilities varying from $P > 0.05$ to $P < 0.001$. It was also demonstrated that the LS means of all subprimal cuts from the legs (topside, silverside, thickflank and rump) expressed as a percentage of cold carcass weight were higher for carcasses originating from the nn genotype ($P < 0.05$). This could be ascribed to the higher LS means for leg weight ($P < 0.05$) originating from the nn genotype. However, when meat quality traits were compared the shortfall of the nn genotypes became evident. LS means for pH_{45} ($P < 0.001$), pH_{24} ($P < 0.05$), drip loss ($P < 0.001$) and reflectance values ($P < 0.05$) differed and were inferior for this genotype.

The backs and legs were used to prepare cured, smoked bacon and canned hams. Comparison of LS means revealed the following: initial gain in pumped weight was significantly higher ($P < 0.001$) for the NN and Nn genotypes compared to the nn genotypes. Similarly, the total gain in bacon yield (finished product yield) was the highest for the Nn (11.5%) and NN genotypes (10.0%), significantly higher ($P < 0.05$) than the bacon produced from the backs of the nn genotypes (3.4%). Differences between sexes were not significant. Canned hams (without added phosphate) produced from the NN genotypes had a significantly ($P < 0.001$) lower percentage cooking loss (27.92%) compared to that of the Nn (30.12%) and nn genotypes (31.14%), which did not differ. Addition of phosphates (0.3% on final product) had a similar response, with the hams produced from the NN genotype having 13.75% cooking loss, significantly lower ($P < 0.001$) than the Nn (16.87%) and nn genotypes (17.73%).

Results for the two types of fresh sausage manufactured from the meat of the different genotypes (with rusk, without rusk) indicated that for the sausage without rusk that the NN genotypes (15.7%) had lower total moisture losses ($P < 0.05$) compared to the nn genotypes (18.9%), with Nn intermediate (17.4%). The treatment with rusk addition did not differ significantly between genotypes (NN = 12.6%, Nn = 13.0, nn = 14.2%), indicating that some of the disadvantages may be overcome by processing technique. Taste panel evaluation of the fresh sausage made without rusk indicated no differences between genotypes when evaluated for juiciness. In an emulsion type sausage the smoking and cooking losses during manufacturing of the product indicated that the sausage manufactured from the meat of the nn genotype (12.47%) had significantly ($P < 0.05$) higher losses than that from the Nn genotype (11.31%), with NN genotype intermediate (12.35%).

The results from this investigation suggest that the presence of the halothane gene is associated with decreased fresh meat and processing properties (lower water holding capacity, reflected by yield or total loss and higher reflectance values) in certain products. This leads to inferior quality in finished products as well as deminished financial returns for processors.

OPSOMMING

Die karkas-, vleiskwaliteit en prosesseringseienskappe van die drie halotaangenotipes in varke is geëvalueer. Sestig Landras x Grootwit varke (NN = 25, Nn = 19, nn = 16) is op ± 86 kg lewende gewig geslag, die karkasse vir 24 uur verkoel teen 2 °C en sekere karkas- en vleiskwaliteitseienskappe bepaal. Kleinste vierkant (KKG) gemiddeldes bereken vir uitslagpersentasie het aangedui dat die Nn genotipes 'n hoër gemiddelde ($P < 0.05$) het as beide die NN en nn genotipes. KKG gemiddeldes vir karkaslengte was die hoogste ($P < 0.05$) vir die nn genotipes. KKG gemiddeldes vir verskeie mates op die halveerde karkas (mediale vetdiktes) asook vet- en spiermates (oogspierwydte, -diepte, en –oppervlak) lateraal (45 mm vanaf middellyn) van die werwelkolom tussen die 2de en 3de laaste torakswerwel het betekenisvolle bewys gelewer dat die nn genotipe karkasse met 'n hoër maervleisinhoud het as die NN en Nn genotipes. Voorspellingsvergelykings vir enkele van hierdie mates waarmee die KKG gemiddeldes bereken is het bevestig dat die nn genotipes karkasse met minder vet en meer maervleis gee.

Disseksie van geselekteerde snitte (skouers en boude) het ook aangedui dat die KKG gemiddeldes vir been-, vet- en maervleisopbrengs meer voordelig was vir die nn genotipe, met waarskynlikhede wat gewissel het van $P > 0.05$ tot $P < 0.001$. Dit is ook gedemonstreer dat dat die KKG gemiddeldes vir al die maervleissnitte (binneboudstuk, dy, diklies en kruisstuk), bereken as 'n persentasie van koue karkasmassa, hoër ($P < 0.05$) was vir die karkasse afkomstig van die nn genotipe. Dit kan toegeskryf word aan die hoër KKG gemiddelde vir boudmassa ($P < 0.05$) van die nn genotipes. Vergelyking van KKG gemiddeles vir vleiskwaliteitseienskappe het egetr die tekorte van die nn genotipe uitgewys. KKG gemiddeldes vir pH_{45} ($P < 0.001$), pH_{24} ($P < 0.05$), drupverlies ($P < 0.001$) en refleksiewaardes ($P < 0.05$) het verskil en was swakker as die KKG gemiddeldes van die ander genotipes.

Die hamme en lendes afkomstig is gebruik vir die vervaardiging van gepekelde rugspek en geblikte hamme. Aanvanklike gewigstoename, na pekelinspuiting, was betekenisvol hoër ($P < 0.05$) vir die NN (15.2%) en Nn genotipe (14.9%), indien dit vergelyk word met die nn genotipe (8.9%). Soortgelyk was die totale toename in spekopbrengs vir die NN (10%) en Nn genotipe (11.5%) betekenisvol hoër ($P < 0.05$) indien dit vergelyk word met die nn genotipe (3.4%). Die geblikte hamme (sonder bygevoegde fosfaat) afkomstig van die NN genotipe het 'n betekenisvol laer ($P < 0.001$) persentasie kookverlies (27.92%) gehad vergeleke met die Nn (30.12%) en nn genotipe (31.14%), wat nie van mekaar verskil het nie. Die byvoeging van fosfate (0.3% in die finale produk) het 'n soortgelyke respons meegebring. Die NN genotipe het betekenisvol ($P < 0.001$) laer kookverliese (13.75%) gehad vergeleke met die Nn (16.87%) en nn genotipes (17.73%).

Die resultate van die twee tipes wors (met graan en sonder graan) wat uit die vleis afkomstig van drie genotipes vervaardig is het aangedui dat, vir die wors vervaardig sonder bygevoegde graan, die NN genotipe betekenisvol ($P < 0.05$) minder vog verloor het (15.7%) vergeleke met die nn genotipe (18.9%). Die Nn genotipe het 'n intermediêre waarde vir vogverlies gehad (17.4%). Die byvoeging van graan het kleiner verkille tussen genotipes (NN = 12.6%, Nn = 13.0%, nn = 14.2%) tot gevolg gehad, met geen betekenisvolle verskille tussen genotipes. Proepaneelevaluering van die wors (vervaardig sonder graan) het geen verskille tussen genotipes ten opsigte van sappigheid uitgelig nie, maar die wors met bygevoegde graan het het wel groter verskille getoon. Die wors vervaardig van die nn genotipe was die sappigste, met die NN genotipe die minste sappig en Nn intermediêr. Vergelyking van berokings- en kookverliese tydens die vervaardiging van 'n emulsieproduk (vienna) afkomstig van die drie genotipes het op 'n betekenisvolle verskil ($P < 0.05$) tussen die Nn (11.31%) en nn genotipe (12.47%) gedui, met NN intermediêr (12.35%).

Die resulate van hierdie studie stel voor dat die teenwoordigheid van die halotaangeen geassosieër is met 'n afname in beide vars vleiskwaliteit en prosseseringseienskappe van sekere produkte (lae aanvanklike pH, bleek kleur, verminderde waterbindingsvermoë, lae finale produkopbrengste). Dit gee aanleiding tot 'n daling in produkkwaliteit en het gevolglik 'n negatiewe finansiële uitwerking op die vleisprosesseerder. Die aanbeveling is dus sterk teen die gebruik van die halotaangeen in enige kommersiële varkproduksiestelsel wat gemik is op die verskaffing van vleisprodukte van hoogstaande gehalte.

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LIST OF ABBREVIATIONS.

ADG	average daily gain
ADP	adenosine diphosphate
AMP	adenosine monophosphate
ATP	adenosine triphosphate
Ca^{2+}	calcium
cDNA	carrier deoxiribonucleic acid
CIE	International Commision on Illumination
CP	creatine phosphate
Cp	Mallow's coefficient
DFD	dark firm dry
FCR	feed conversion ratio
GM	*M. gluteus medius*
HGP	Hennessey Grading Probe
IMP	inosine monophosphate
LMP	lean meat percentage
M	mole
MH	Malignant Hyperthermia
Mg^{2+}	magnesium
MLT	*M. longissimus thoracis*
Na^{+}	sodium
NN	homozygous halothane negative genotype
Nn	heterozygous halothane negative genotype
nn	homozygous halothane positive genotype
p	parameters used in regression analysis
P	Probability
pH_1	pH measured after 1 h
pH_{24}	pH measured after 24 h
pH_{45}	pH measured after 45 min
pH_i	initial pH
pH_u	ultimate pH
P_I	inorganic orthophosphate
PCR	polymerase chain reaction
PSE	pale, soft and exudative

PSS	Porcine Stress Syndrome
RNA	ribonucleic acid
SR	sarcoplasmic reticulum
SS	sum of squares
STPP	sodium tripolyphosphate
T_1	1st thoracic vertebra
T_{2-3}	2nd–3rd last thoracic vertebrae
T_{2-3}-45mm	2nd–3rd last thoracic vertebrae, 45 mm from the midline
WHC	water holding capacity

CONTENTS

1	INTRODUCTION	1
2	A REVIEW OF THE STRUCTURE AND COMPOSITION OF MAMMALIAN SKELETAL MUSCLE	4
2.1	Introduction	4
2.2	Morphology	5
2.3	Muscular contraction *in vivo*	7
3	POST MORTEM CHANGES IN SKELETAL MUSCLE	8
3.1	Introduction	8
3.2	Changes in energy-supplying compounds and substrates post mortem	8
3.3	pH changes in the post mortem muscle	10
3.4	The effect of stress on post mortem glycolysis	11
3.5	The effect of temperature on post mortem glycolysis	11
3.6	Physical changes in post mortem muscle	12
4	CLASSIFICATION OF MUSCLE FIBRE TYPES	13
4.1	General	13
4.2	Effect of exercise, breed and anatomical position of muscle on fibre characteristics	14
4.3	Giant fibres	15
5	MALIGNANT HYPERTHERMIA AND PORCINE STRESS SYNDROME	16
5.1	General	16
5.2	Aetiology of MH	16
5.3	PSS, MH and Ca^{2+} regulation in the muscle	17
5.4	Halotyping	17
6	PRESLAUGHTER HANDLING AND MEAT QUALITY	20
6.1	General	20
6.2	Feeding	21
6.3	Transportation and lairage	21
6.4	Stunning	24
7	CARCASS AND MEAT QUALITY CHARACTERISTICS	26
7.1	General	26
7.1.1	Midline measurements	26
7.1.2	Lateral measurements	27

7.1.3	Cut distribution and carcass composition	28
7.2	Meat quality	29
7.2.1	Initial and ultimate pH	30
7.2.2	Colour	31
7.2.3	Drip loss	32
7.2.4	Chemical composition and eating quality	32
7.3	Conclusion	33
8	PROCESSED PORK PRODUCTS	34
8.1	General	34
8.2	Cured products	34
8.3	Meat quality and the acid/base status of the pig	35
8.4	The role of salt and various phosphates on product quality	35
8.4.1	Phosphates	36
8.4.2	Salt	37
8.5	Moisture retention in cured products: the use of other additives	38
8.6	Cured meat colour and phosphates	38
8.7	Meat quality and shrinkage during transporting and processing	39
8.8	Tumbling and massaging	39
8.9	The effect of heating rate and pH on thermal gelation	40
8.10	Fresh comminuted and emulsion meat products	40
8.11	The effect of salt, fat and pH on sensory characteristics of emulsified meat products	41
8.12	The role of particle size on sensory and physical properties of comminuted products	42
8.13	Connective tissue and phosphates: effect on emulsion products	42
8.14	Conclusion	42
9	MATERIALS AND METHODS	43
9.1	Pigs	43
9.2	Genotype identification	43
9.3	Slaughter procedures	43
9.3.1	Dressing percentage	44
9.3.2	Chilling loss	44
9.4	Carcass measurements	44
9.4.1	General remarks	44
9.4.2	Cutting and splitting of carcasses	45

9.4.3	Removal of the three primal cuts	45
9.4.3.1	Leg	45
9.4.3.2	Shoulder	45
9.4.3.3	Back	46
9.4.4	Deboning technique	46
9.4.4.1	Leg	46
9.4.4.2	Shoulder	47
9.4.4.3	Back	47
9.5	Linear measurements	52
9.5.1	Carcass length	52
9.5.2	Length:live weight ratio	52
9.5.3	Midline fat measurements	52
9.5.4	Percentage predicted lean, fat thickness and muscle depth measurements	52
9.5.5	Muscle area	53
9.6	Meat quality	53
9.6.1	pH	53
9.6.2	Drip loss	54
9.6.3	Colour measurements	54
9.6.4	Cooking loss and shear value	54
9.6.5	Chemical analyses	55
9.7	Manufacturing techniques	55
9.7.1	Preparation of back bacon	55
9.7.2	Preparation of comminuted products	56
9.7.3	Canned hams	57
9.7.4	Fresh pork sausage	57
9.7.5	Viennas	58
9.8	Sensory evaluation	59
9.9	Statistical analysis	61
9.9.1	Null hypothesis and test of significance	61
9.9.2	Correlation	61
9.9.3	Multiple linear regression	62
9.9.4	Analyses of ordinal data	63
10	RESULTS AND DISCUSSION	64
10.1	Carcass quality	64
10.2	Carcass weights	64

10.3	Midline carcass measurements	64
10.4	Lateral carcass measurements and predicted lean yield	68
10.5	Primal cut distribution and cutting yields.	70
10.5.1	Shoulder yield	70
10.5.2	Leg yield	76
10.6	Meat quality	82
10.6.1	pH (pH_{45} and pH_{24})	82
10.6.2	Chilling and drip loss	86
10.6.3	Meat colour	87
10.6.4	Cooking loss and meat tenderness	88
10.6.5	Chemical composition	89
10.6.6	Conclusion	89
10.7	Processed products	91
10.7.1	Back bacon	91
10.7.2	Canned hams	94
10.7.3	Fresh pork sausage	97
10.7.3.1	Processing losses of fresh pork sausages	97
10.7.3.2	Sensory evaluation of fresh pork sausages	99
10.7.4	Viennas	102
10.7.5	Summary	102
10.8	CORRELATION AND REGRESSIONS	104
10.8.1	Selected carcass yields: shoulder	104
10.8.2	Selected carcass yield: leg	105
10.8.3	Meat quality	106
10.8.4	Final bacon yield	107
10.8.5	Conclusion	107
11	DEVELOPMENT OF OBJECTIVE PROCEDURES TO IDENTIFY PSE PORK AND PREDICT PROCESSED PORK QUALITY	109
12	CONCLUSIONS	112
12.1	Carcass quality characteristics	112
12.2	Meat quality characteristics	113
12.3	Processing characteristics	114
Annex A		116
Annex B		119
REFERENCES		139

1. INTRODUCTION

Meat processing is an ancient technique that probably began when early man first realised that salt was an effective way of preserving and extending the keeping quality of meat. The ancient Egyptians salted and sun dried meat, whereas the Romans were one of the first civilisations to use ice and snow as a means of preserving food. One of the results of the Industrial Revolution was the concentration of the population in urban areas which brought about a much greater demand for food, since these urbanised families produced less of the food that they required. Together with these changes came an improvement in the social and economic status of the industrial worker with the consequent rise in their consumption of meat, vegetables, dairy products and fruit and a decreased cereal (bread) consumption. This gradual change in eating habits also created a rising desire, as well as a demand, for food with better keeping qualities and improved taste. Rising labour costs also prompted the rapid development of processed foods. After about 1850 great advances were made in commercial food processing, especially in meat processing, with its initial aim being the prevention of product spoilage. However, meat processors soon realised that these processes also added value to the product in terms of flavour, aroma, colour and nutritive value (Encyclopaedia Britannica, 1990).

Agriculture, and more specifically the animal sciences, developed along a similar pattern. In an effort to satisfy consumer demands the application of scientific principles to the selection of 'superior' breeding animals, with increased efficiencies for production purposes, were required. Gradually consumers have come to prefer more lean tissue and less fat in meat, and thus, starting in the 1950's, the muscular meat type pigs were developed over several decades of intense selection and crossbreeding. However, these selection and breeding principles for increased lean yield and improved feed conversion ratios (FCR) in pigs also resulted in less desirable associated characteristics. One of the most important of these less desirable characteristics is the condition known as pale, soft and exudative (PSE) pork. This type of meat is pale in colour, has a soft texture and exudes a high quantity of moisture compared to normal pork. There is normally also very little marbling fat present in muscles exhibiting this condition, and the muscles have a tendency to be disassociated from the connective tissue attaching the subcutaneous fat layers. The PSE condition is most commonly caused by some form of stress suffered prior to slaughtering, which results in rapid glucogenolysis and a concomitant accumulation of lactic acid in the muscle tissues. After slaughtering, the accumulation of lactic acid continues and can be witnessed by a rapid drop in muscle pH, which, together with high post mortem temperatures, results in the characteristic PSE type pork. When used for products such as sausages and smoked or canned meats, PSE pork, because of its poor water binding ability, results in inferior end-products. Smoked products from

PSE pork also tend to shrink appreciably more than those made from normal pork. The finished products, whether smoked or comminuted, will usually be lighter in colour compared to normal pork. PSE pork is also not desired in the manufacturing of comminuted meat products, because of its poor water binding and fat emulsifying abilities.

PSE has been linked to a genetic mutation in pigs, known as Malignant Hyperthermia (MH). MH is a pharmacogenic disease which can be triggered by drugs such as halothane, as well as numerous natural stressors (transport, mating, fighting), and is characterised by symptoms such as muscle rigidity, increased rectal temperature, increased cardiac output, hyperkalemia and respiratory and metabolic acidosis (Mitchell and Heffron, 1982). These physiological and biochemical changes are invariably fatal. The MH gene is an outosomal recessive gene (also known as the halothane gene) which was probably inadvertently selected for as it has a high genetic correlation with certain advantageous characteristics such as increased growth, better FCR and a higher proportion lean in the carcass. These characteristics are all interdependent on one another, since muscle is an early maturing tissue, FCR is higher in young animals, and growth rate is higher before puberty than thereafter. However, these advantages are offset by the less desirable trait (PSE) which characterises the meat from pigs with the MH gene. It must be pointed out that the PSE condition is not limited to genotypes that have the MH gene, but the incidence of PSE is much higher in these animals.

The development of a non-invasive test to determine halothane genotype [homozygous (NN) and heterozygous (Nn) non-reactors, and the homozygous (nn) reactors] added a lot of impetus in determining the effect of the gene on certain growth, carcass and meat quality characteristics. In the past this was not possible since the traditional tests (such as the halothane challenge test) that were available could only differentiate between phenotypes (reactors and non-reactors), and were not able to identify the heterozygous (Nn) from the homozygous (NN) non-reactors. The non invasive test is based on the assumption that a single point mutation in the porcine gene for the skeletal ryanodine receptor (*ryr 1*) is the causal factor for MH. Five of the major breeds consisting of populations of predominantly lean, heavily muscled pigs (Pietrain, Yorkshire, Landrace, Poland China and Duroc) tested, showed a strong correlation between this mutation and MH. This suggests that the mutation either has a common source for all these breeds or that it has been a recurring mutation selected for in each of these breeds. On the basis of the above mentioned, researchers have concluded that a common ancestry for all the MH animals in the five pig breeds is indicated.

The purpose of this investigation can be broadly defined as the determination of certain carcass and meat quality characteristics and processing properties of the different genotypes, including subjective and sensory techniques. Based on these results, a recommendation will then be made regarding the genotype, or genotypes, most suitable for production and processing purposes that will meet the demands of pork producers, pork processors and pork consumers.

2. A REVIEW OF THE STRUCTURE AND COMPOSITION OF MAMMALIAN SKELETAL MUSCLE

2.1 Introduction

Muscle is generally divided into three types, namely skeletal, smooth and cardiac muscle. Skeletal muscle comprises the greater mass of the somatic musculature and is the primary source of meat derived from a carcass and will thus be the main focus of this review. As a result of the physical demands placed on it, skeletal muscles exhibit a large variation in size, shape, attachment, colour, blood and nerve supply and in their association with other tissues (Davey & Winger, 1988; Lawrie, 1991). Nonetheless, all muscles share a similar basic structural pattern (Fig. 2.1.1).

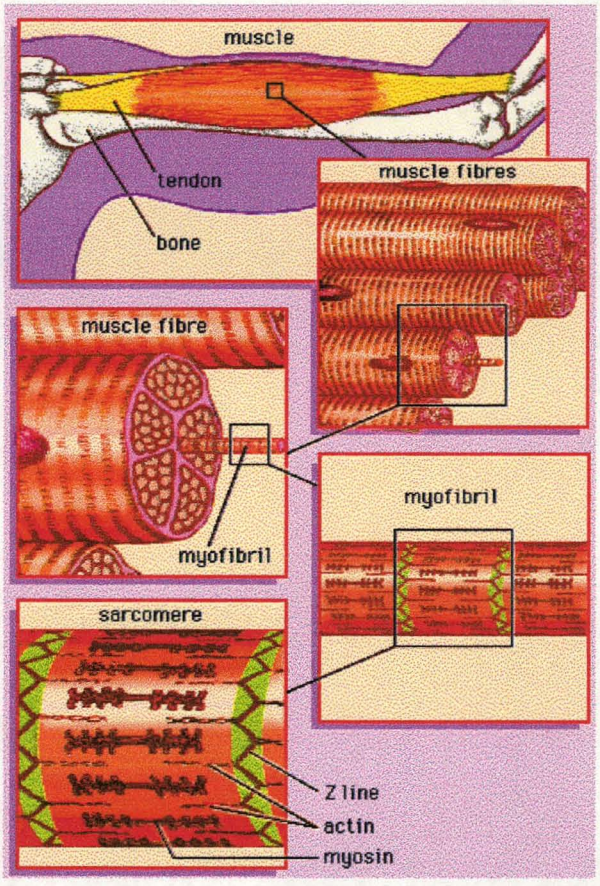

Figure 2.1.1 Schematic representation of the basic skeletal muscle structure (Encyclopaedia Britannica, 1998)

Each muscle is surrounded by a sheath of connective tissue known as the epimysium. From the inner surface of the epimysium septa of connective tissue, known as the perimysium, penetrate the muscle, separating the muscle fibres into bundles. A fine connective tissue framework originating from the perimysium, called the endomysium, passes further inward and surrounds each individual muscle fibre (Lawrie, 1991). The size of the muscle fibre bundles determine the texture of the

muscle (Walls, 1960). Muscles that control fine movements, such as those of the eye, have a fine texture, whereas those performing grosser movements are coarse in texture. However, the proportion of connective tissue is higher in muscles that are involved in fine movements. The relative proportions of connective tissue and muscle fibres vary between muscles, this phenomenon partially accounts for the differences in relative toughness of different muscles (Lawrie, 1991).

2.2 Morphology

Skeletal muscle is made up of individual muscle fibres, which are the basic cellular units of living muscle and of meat. All skeletal muscle originates and inserts on the skeleton in aggregates of connective tissue or tendons, and the muscle fibres within the muscle bundles are arranged parallel and in series between the tendinous ends so that the force of contraction of the units is additive (Ganong, 1975). Muscle fibres are unusual cells as they are long, multi-nucleated and cylindrical in shape. Muscle fibres can attain a length of 34 cm, although they are only 10-100 μm in diameter (Walls, 1960). Factors such as species, breed and sex can cause a variation in fibre diameter (Joubert, 1956).

The muscle fibres are made up of fibrils, which have the same diameter irrespective of size or development of the fibre (Davies, 1989). As noted, each fibre is surrounded by the endomysium, which in turn surrounds a sheath, the sarcolemma. Approximately 80% of the muscle cell volume is made up of threadlike contractile cylinders called myofibrils (Fig.2.2.1). The myofibrils are surrounded by a fluid phase, the sarcoplasm, which contains; cell components (such as the nuclei), responsible for cell differentiation and morphology; the sarcoplasmic reticulum (SR), responsible for nervous impulse transmission; the mitochondria, responsible for oxidative metabolism; the supporting cytoskeletal framework and glycogen granules, which serve as a reserve of metabolic energy.

Under magnification the fibres show well developed transverse striations (Fig. 2.2.1). If samples from stretched and contracted muscles are compared, the striations will appear relatively far apart in the stretched muscle and closer together in the contracted muscle. The transverse striations on muscle fibres are due to the precise alignment of A-bands (anisotropic bands) and I-bands (isotropic bands) on fibrils within the fibre. In most stained preparations for light and electron microscopes, A-bands appear darker than I-bands (the reverse appearance to that seen with polarized light). This is because the birefringent A-bands contain a greater density of protein. A-bands also appear darker than I-bands when unstained preparations are observed with a phase contrast light microscope (a special type of microscope that shadows differences in glassy properties of the specimen). The light

I-band is divided by the dark Z-line, and the dark A-band has the lighter H-band in its centre. The H-band is in turn bisected by dark M-lines, and this line plus the narrow lighter coloured areas on either side of these areas are called the pseudo-H zones. The repeating unit of a regular series of transverse striations is termed the sarcomere, and a structural unit is usually considered to be from Z-line to Z-line. The Z-line resembles a woven disk, like the bottom of a wicker basket, and it extends as a partition across the fibril. At an ultrastructural level, the sarcomere consists of two sets of filaments (thick and thin filaments) running parallel to the muscle (Squire, 1981).

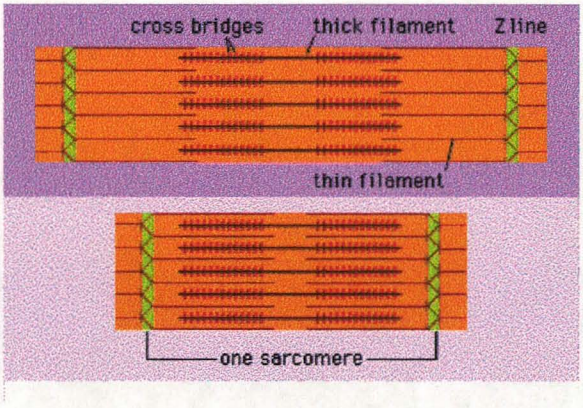

Figure 2.2.1 Schematic representation of the contractile unit of skeletal muscle (Encyclopaedia Britannica, 1998)

The thick filaments (12 nm in diameter, 1500 nm in length), called the myosin filaments, are twice the diameter of the thin filaments, and are made up of myosin; the thin filaments (called the actin filaments) are made up of actin, tropomyosin and troponin. The thick myosin filaments form the A-bands, whereas the lighter I-bands are formed by the thin actin filaments (both actin and myosin form part of the myofibrillar proteins). When the muscle is in a relaxed state, the actin and myosin filaments do not overlap, evident as the lighter H-bands in the centre of the A-bands. The Z-line, which binds the sarcomere at each end, transects the fibrils and connect to the actin filaments. The pseudo-H zone is created by the symmetrical arrangement of the myosin molecules on either side of the centre of the sarcomere. The M-line is due to a central bulge in each of the thick filaments. At these points slender cross connections are found which hold the myosin filaments in proper array. Electron microscopic examination of a transverse section through the A-band shows that each myosin filament is surrounded by 6 actin filaments in a regular hexagonal pattern. When a muscle fibre contracts, the thick filaments slide between the thin filaments so that the I-band gets shorter. However, the length of the A-band remains constant. This is called the sliding filament theory of muscle contraction. Electron microscopy also indicates that the individual myosin molecules have

enlarged heads that form cross-linkages with other myosin heads, which is also the site for the interaction with ATP and the actin molecules during muscle contraction.

The actin filaments consists of two helical wound strands composed of globular sub-units which form a double helix (Hanson & Lowy, 1963). Tropomyosin molecules, which are minor myofibrillar proteins, are long filaments that run along each side of the actin polymers. Located at regular intervals (38.5 nm) on the filaments, is another protein, troponin, which consists of three units, T (tropomyosin binding), C (Ca^{2+} binding) and I (inhibitory effect on actin-activated myosin ATPase activity). Each thin filament contains 300-400 actin molecules and 40-60 tropomyosin molecules. The myosin filaments represent the lateral aggregation of the individual tadpole-like molecules of myosin, and is comprised of approximately 200 myosin molecules (Huxley, 1963). The myosin molecules aggregate with the tails towards one another and the heads directed towards the Z line, thus forming a cylinder (with tapering ends) about 1.5 μm in length.

2.3 Muscular contraction *in vivo*

Muscular contraction in the living animal is characterized by the sliding action of the thick and thin filaments of the sarcomere (Squire, 1981) in the presence of sufficient adenosine triphosphate (ATP). Activation of the muscle is initiated by a nerve stimulus arriving at the motor end plate, which causes a reverse in the polarization of the sarcolemma. The repolarization is dispersed through the transverse tubule (T-tubule) system of the muscle fibre and induces depolarization of the SR. This causes the dissociation of Ca^{2+} ions from calsequestrin (an acidic protein by which they are normally bound in the sarcotubular system) and the release of Ca^{2+} from the SR into the sarcoplasma and resulting in a Ca^{2+} concentration increase from approximately 0.10 μM to 10 μM (Lawrie, 1991). This triggers the cyclical cross bridging between thick and thin filaments in the region of the overlap of the thick and thin filaments (Smellie, 1974). Troponin C on the tropomyosin strands is saturated by Ca^{2+} and causes a configurational change in Troponin I, which then no longer prevents the interaction of actin with MgATP. The contractile ATPase is activated, providing energy for the contraction by dephosphorylating MgATP to MgADP and energy. The energy provided by the dephosphorylation is used to pull the actin filament towards the centre of the sarcomere. The MgADP on the myosin is rephosphorylated to MgATP by either a direct exchange with cytoplasmic ATP, by the action of ATP:phosphotransferase or ATP:AMP phosphotransferase. This process will continue as long as sufficient ATP is supplied, or until the SR retrieves the released Ca^{2+}. With the cessation of contraction, Ca^{2+} is recaptured by the calcium pump, which depends on ATP for energy. Troponin C and I, no longer saturated by Ca^{2+}, return to their resting configuration, thus preventing actin:myosin interaction (Lawrie, 1991).

3. POST MORTEM CHANGES IN SKELETAL MUSCLE

3.1 Introduction

After slaughter, the body tissues are deprived of oxygen, and energy is by anaerobic glycolysis only (Fig 3.1.1). The muscles, however, are still biochemically active post mortem and will utilise glycogen and high energy compounds in an attempt to maintain cellular homeostasis until rigor mortis is irreversibly established. The physical and biochemical post mortem changes have been extensively reviewed by Bendall (1979).

1 $ATP \rightleftharpoons ADP + P_i$

2 creatine-P + ADP ⇌ creatine + ATP

3 **Under aerobic conditions:**

$(glycogen)_n + 9ADP + 9P_i \rightarrow (glycogen)_{n-1} + 9ATP + 2\ pyruvate \rightarrow 2\ pyruvate + 30ADP + 3P_i - 6CO^2 + 30ATP$

4 **Under anaerobic conditions:**

$(glycogen)_n + 3ADP + 3P_i \rightarrow (glycogen)_{n-1} + 3ATP + 2\ pyruvate \rightarrow 2\ pyruvate \rightarrow 2\ lactate$

5 $2ADP \rightarrow ATP + AMP$

6 $AMP \rightarrow IMP + NH_3$

Figure 3.1.1 Chemical changes in muscle during aerobic and anaerobic metabolism (Barton-Gade *et al.*, 1988)

3.2 Changes in energy-supplying compounds and substrates post mortem

Glycogen is utilised in the skeletal muscle as the substrate for glycolysis. It is the primary storage carbohydrate in muscle fibres and is located as single granules or lumps in the sarcoplasm between myofibrils and under the plasma membrane of the fibre (Swatland, 1995). Glycogen, a polysaccharide formed by the linking together of large numbers of glucose units, normally comprises about 1 % of the muscle weight (Lawrie, 1979). Pig muscle shows a rapid depletion of glycogen, reaching low levels within 3 to 5 h after death (Sayre *et al.*, 1963a, b; Beecher *et al.*, 1965a, b; Bodwell *et al.*, 1966). The amount of glycogen remaining when the muscle goes into rigor mortis lies in the range of 2 to 30 % of resting levels and depends on the original resting concentration and the rate at which the glycolytic enzymes become inactive. The development of a low pH as a result of lactic acid production during anaerobic glycolysis is a major factor in the inactivation of phosphorylase and the other glycolytic enzymes.

The concentration of ATP in the resting, living muscle ranges from between 5.7 and 8.1 µmol/g (Bendall, 1973), depending on the species or the specific muscle. In resting muscle, ATP is slowly dephosphorylated to ADP, producing energy for a wide range of metabolic requirements. The dephosphorylation of ATP is continued after death and, if not replenished, would lead to a total absence of this potential energy source. However, the rate of post mortem replenishment of ATP is less than the rate of ATP breakdown due to the inefficient system of ATP resynthesis via anaerobic metabolism. For example, ATP levels in beef muscle drop to about 17 % of its initial (live) value at 48 h post mortem (Bodwell *et al.*, 1965). Thus, after an initial delay, the ATP concentration level is lowered and the muscle enters a rapid phase of rigor development, which continues until all the ATP is depleted. After the disappearance of ATP, which, when present, acts as a plastisizer and prevents the formation of cross bridges (Lawrie, 1991), actin-myosin cross bridges are irreversibly formed and the muscle thus enters a state of inextensibility or rigor mortis.

During the early delayed phase of rigor development, ATP is resynthesized through the transfer of phosphate from creatine phosphate (CP) and, to a lesser degree, through glycolysis. During this phase the thick and thin filaments slide past one another with the resultant extensibility and contraction upon stimulation. This scenario remains as long as the CP stores last and ATP can be resynthesized efficiently. When the CP stores are depleted, resynthesis of ATP depends largely on glycolysis, which signals the second or rapid stage of rigor development.

This rapid phase is characterized by a drop in muscle ATP concentration which cannot be sufficiently replenished by anaerobic glycolysis. Cross bridges between actin and myosin form in the region of the overlap between the thick and thin filaments. The ADP, which is the product of ATP dephosphorylation, is converted to both AMP and ATP by the phosphotransferase enzyme myokinase. For every 2 molecules of ADP used, a molecule of AMP and ATP is formed. The AMP is then irreversibly deaminated to inosine monophosphate and ammonia (Fig. 3.1.1).

The time involved in the development of rigor mortis in rested and fed animals show a wide variation with regard to factors such as amount of death struggle, post-slaughter handling, internal temperature and location of the skeletal muscle (Davey & Winger, 1979). The excised *M. longissimus thoracis* (MLT) of oxen reach rigor mortis in approximately 24 h at 7°C, 16 h at 17°C and 5 h at 37°C. Various muscles within species also show differing times in reaching full rigor mortis. In chilled beef carcasses the *M. psoas major* enters rigor mortis after approximately 8 h, whereas the MLT takes about 24 h.

3.3 pH changes in the post mortem muscle

Resting pH values in living muscle vary between muscles and species of farm animals from 7.08 to 7.40 (Bate-Smith, 1948; Bendall, 1973). Factors such as animal species, muscle type and stress associated with slaughter will also have an effect on post mortem pH decline patterns. Beef muscle pH normally falls to a range of 6.9 to 7.0 within 10 min post mortem, and then declines to about 5.5 - 5.6 at 48 h post mortem (Bodwell *et al.*, 1965; Cassens & Newbold, 1966). Pearson *et al.* (1973a, b) reported mutton muscle pH values of about 6.9 - 7.0 in less than 30 min post mortem. Marsh and Thompson (1958) reported similar results for lamb muscle 30 - 40 min post mortem. Hallund and Bendall (1965) reported pork muscle pH at 10 - 15 min post mortem to be 6.6 - 6.8.

These results suggest that the pH for the MLT muscle declines curvilinearly from an initial value until its final 48 h pH of about 5.4 - 5.7 (Greaser, 1986), with pork muscles showing an inherent faster pH decline than that of beef and mutton. Lawrie (1979) reported that the drop in muscle pH is faster in pork MLT when compared to beef MLT, which is intermediate, and slowest in horse MLT. The most notable differences between species occur during the early post mortem period (first 3 h). Different muscles from the same species also have different post mortem curves (Lawrie, 1979). For example, in pork the *M. psoas*, *M. semimembranosus* and MLT have distinctly different curves. The MLT and *M. semimembranosus* reached pH 5.8 at 3.7 and 5.4 h, respectively, with the *M. psoas* reaching pH 5.8 at 2.5 h post mortem. The distance of sampling from the muscle surface may also influence pH readings with measurements taken deep in the muscle showing a noticeably faster decline in pH than the samples taken closer to the muscle surface. Tarrant (1981) indicated that beef *semimembranosus* muscle requires 24 - 48 h to reach an ultimate pH at 1.5 cm from the surface, but needs only 12 h and 6 h at 6 and 8 cm, respectively.

The decrease in pH is also accelerated by mincing or grinding (Newbold & Scopes, 1971). Ground lamb muscle resulted in a pH decrease from 6.82 to 5.63 within 6 h, compared to a decrease in pH to 6.46 for the unground controls (Pearson *et al.*, 1973b). The addition of calcium and epinephrine accelerates the pH drop over that of mincing alone, although the decline appears to be associated with some mechanism other than the conversion of phosphorylase b (inactive) to phosphorylase a (active). Newbold and Small (1985), in accordance with these results, found that conversion of phosphorylase b to a is only transitory in nature and probably does not significantly contribute to the rapid pH decline that results from electrical stimulation.

3.4 The effect of stress on post mortem glycolysis

Preslaughter stress greatly affects the levels of high energy phosphates in the muscle. The PSE and dark, firm and dry (DFD) conditions in pig muscles are related to preslaughter stress and is a consequence of the alteration in the levels of high energy phosphates and their resultant metabolites (Briskey, 1964; Sybesma & Eikelenboom, 1978). Fast glycolyzing muscles are present in pig and bovine muscles, although less frequent in the latter (Fischer & Hamm, 1980).

Results by Fischer and Hamm (1980) demonstrated that ATP and CP levels in fast glycolyzing muscles decrease due to a combination of both rapid glycolysis and glycolysis occurring earlier post mortem than in normal muscle. Both these compounds (ATP and CP) were present in the fast glycolyzing muscles at lower initial concentrations, but after 1 h post mortem CP was almost absent with low ATP levels, indicating a rapid utilisation of the high energy phosphates. The lower initial pH values were associated with lower muscle glycogen levels, lower water holding capacity (WHC), paler colour and higher concentrations of lactic acid when compared to normal muscles. Comparison of species indicate that the effect of low pH on beef muscles are, however, less severe than in the case of pork muscles (Fischer & Hamm, 1980).

The connection between stress, meat quality and the rate of glycolysis has been well documented. Stress greatly accelerates the rate of glycolysis and contributes to the development of PSE muscle (Briskey *et al.*, 1959; Briskey, 1964; Sair *et al.*, 1970). Callow (1936) reported that the depletion of glycogen stores, caused by stress, was a contributing factor to the development of bacon spoilage, due to the high ultimate pH and reduced shelf life in DFD (or potential DFD) muscles.

3.5 The effect of temperature on post mortem glycolysis

Post mortem glycolysis is significantly affected by muscle temperature (Marsh, 1954). High post mortem temperatures cause an accelerated drop in pH (used as an indicator of glycolysis) whereas lower temperatures retards the rate of glycolysis. Results from Bodwell *et al.* (1966) showed that pork MLT held at -29°C for 3 h resulted in a pH decline from 6.54 to only 6.24, whereas MLT held at 38°C for 3 h rapidly declined to pH 5.48. Thus, high post mortem temperatures caused an accelerated glycolysis, whereas low temperatures delayed the glycolytic effect. Full rigor, at both temperatures (23°C and 38°C), were however, reached 48 h post mortem. Cold shortening (the shortening of pre-rigor muscle if exposed to chilling temperatures of about 10°C) occurs in beef, sheep and turkeys, but only in pigs under experimental conditions or ultra rapid chilling, but with considerably less intensity (Bendall, 1975). However, in chickens cold shortening is insignificant (Lee & Rickansrud, 1978).

3.6 Physical changes in post mortem muscle

At the time of death skeletal muscles are flaccid and highly extensible. However, within a few hours the muscles become inextensible and relatively rigid, resulting in the condition known as rigor mortis. The time of onset of rigor mortis can be influenced by a number of factors such as temperature or struggling at the time of death. Higher temperatures and struggling speed up glycolysis and hasten the onset of rigor mortis due to an accelerated depletion of metabolic energy compounds such as glycogen. In such animals the time course of rigor development is brief, being restricted by an early cessation of glycolysis (Bendall, 1973). Stimulation of the respiratory system accelerates aerobic metabolism and delays rigor development (Lawrie, 1979). Thin strips of muscle exposed to the atmosphere produces ATP so efficiently that rigor mortis can be delayed and CP resynthesized to levels above its original concentration.

Early work done on the effect of rigor mortis on meat tenderness resulted in conflicting conclusions. Paul *et al.* (1944) showed that delayed cooking after slaughter resulted in more tender meat than cooking immediately after slaughter. Ramsbottom *et al.* (1945) reported that beef steaks fried in deep fat were more tender if cooked immediately after slaughter and increased in toughness on delaying cooking for 24 - 48 h. Paul *et al.* (1952) reported that steaks fried in deep fat within 1 h of death had the lowest shear force values and reached maximum shear force readings (when cooked) 24 - 48 h post mortem, after which tenderness increased on subsequent storage. Large roasts cooked to an internal temperature of 63°C were the toughest when cooked immediately, with decreasing shear force values as cooking was delayed. These results suggest that prerigor meat is tender, and, if cooked in thin pieces at high heat so that rapid heat penetration is achieved, it is even more tender, as suggested by Ramsbottom *et al.* (1945). With large roasts, heat penetration is delayed and the muscle goes into rigor mortis before cooking is completed, resulting in tough meat.

4 CLASSIFICATION OF MUSCLE FIBRE TYPES

4.1 General

Muscles can be broadly classified as 'red' or 'white' based on fibre type (Needham, 1926). 'White' muscles have a predominantly anaerobic metabolism and are more susceptible to the development of PSE meat than 'red' muscles, which have a predominantly aerobic metabolism (Moody and Cassens, 1968). Examples of both muscles types are given in Table 4.1.1.

Table 4.1.1 Muscles grouped according to colour classification.

Predominantly white muscle (anaerobic metabolism)	Predominantly red muscle (aerobic metabolism)
M. longissimus thoracis	*M. semispinalis*
M. semimembranosus	*M. serratus ventralis*
M. biceps femoris	*M. quadriceps femoris*
M. gluteus medius	

However, fibre typing is not always clear cut and a certain amount of diversity does exist. Moody and Cassens (1968) used colour to differentiate between different fibre types, while Barnard *et al.* (1971) used differing contractile and metabolic characteristics. Cassens and Cooper (1971) reviewed the commonly used methods to classify fibre types (Table 4.1.2).

Table 4.1.2 Fibre types grouped according to physical, chemical and morphological characteristics.

Colour	Twitch	ATPase	Energy supp.	Mitochondria
Red	Slow	I	Oxidative	Abundant
	Fast	IIA	Oxidative	Intermediate
White	Fast	IIB	Glycolytic	Limited

Type I fibres (based on ATPase activity) are slow contracting, have a high oxidative potential and are rich in mitochondria. Red fibres have a more abundant blood supply and contain more ribonucleic acid (RNA), whereas type II fibres are faster contracting and have a higher oxidative potential than type I fibres. These fibres have, compared to red fibres, narrower Z-lines, a smaller blood supply and a better developed SR, which is apparently related to their faster rates of contraction and relaxation (Romanul, 1965; Gauthier, 1970; Schiaffino *et al.*, 1970; Burke *et al.*,

1971; Wiles *et al.*, 1979; Saltin & Gollnik, 1983). Type IIA fibres mostly have a much higher oxidative capacity compared to type IIB. The later seem to be highly glycolytic (Brooke & Kaiser, 1970). The histochemical stain for glycogen, although only semiquantitative, is useful for determining glycogen utilisation by the different fibre types during different levels of activity. Type I fibres are usually involved in low to moderate activities, whereas type II fibres are used for high intensity work (Gollnick *et al.*, 1974).

4.2 Effect of exercise, breed and anatomical position of muscle on fibre characteristics

Muscles used to maintain posture, as well as the more frequently used muscles, have a higher proportion of oxidative or type I fibres when compared to muscles less regularly used (Kiessling & Hansson, 1983). In comparison to other mammalian muscles, pig muscles generally have a lower percentage of type I fibres and are therefore lighter in colour. Moderate exercise increases the oxidative capacity of muscles in pigs, but with little improvement in meat colour or WHC (Essén-Gustavsson *et al.*, 1988; Hansson *et al.*, 1991). These researchers concluded that much heavier exercise may be necessary to produce desirable effects on carcass lean:fat ratio and meat quality characteristics, such as colour. However, this is not a commercially realistic approach.

Fibre composition, area and metabolic profile does show remarkable differences within and between pig breeds (Rahelic & Puac, 1981; Monin *et al.*, 1987). Compared to domesticated pigs, wild pigs have a higher oxidative capacity as well as a higher type IIA:IIB fibre ratio (Solomon & West, 1985). Fibre type composition and capillary:fibre ratio are similar in domesticated stress susceptible and stress resistant pig breeds, but on cross section, fibre areas were shown to be larger and capillarisation (cap/mm^2) lower in the case of the stress susceptible pigs (Cooper *et al.*, 1969; Essén-Gustavsson *et al.*, 1992). The lower cappilarisation observed in the stress susceptible pigs was probably due to the low pH, which, if close to pH 5.5, will be at the iso-electric point of the principal muscle proteins. This will cause a decrease in interfilament spacing and thus a decrease in WHC, with a resultant increase in drip loss.

Comparison of different muscles show that the MLT has a large proportion of glycolytic (type IIB) fibres (80-90%) whereas the *M. vastus intermedius* has predominantly oxidative (type I) fibres (70-80%). A characteristic readily observed in porcine muscles is the clustering of red (type I) and white (type IIA and IIB) fibres into groups within a muscle, which is in contrast to the more commonly found checkerboard mode seen in other species (Cassens & Cooper, 1971). Depending on their position, fibres show a marked variation in oxidative capacity. Type IIB fibres, in the vicinity of type I and type IIA fibres, display a higher oxidative capacity compared to type IIB

fibres close to the fascicular border. The metabolic profile can also differ in the *M. gluteus* and MLT of different breeds of pigs (Hampshire, Yorkshire, Swedish Landrace) which, nevertheless, have similar fibre type compositions in each muscle (Essén-Gustavsson & Fjelkner-Modig, 1985).

4.3 Giant fibres

At the end of the 1960's a new type of muscle fibre was described. These fibres are particularly prevalent in the musculature of stress susceptible pigs and are found predominantly in the borders of fascicles among type II fibres (Cooper *et al.*, 1969). They are called 'giant fibres' due to their increased size. Biochemically, these fibres are intermediate between red and white fibres with a low amylophosphorylase activity and high ATPase activity (Lawrie, 1984). It has been suggested that giant fibres are a phenomenon produced post mortem. However this may be doubtful since giant fibres were found in various animal species from which muscles were obtained after various killing procedures (Schmitt & Dumont, 1979). Administration of porcine somatotropin has shown an increase in fibre area in all fibre types in the MLT, however, giant fibres were only observed in some of the porcine somatotropin treated pigs, and not in the untreated controls (Solomon *et al.*, 1990).

Apart from a genetic predisposition to develop PSE due to certain environmental conditions, muscle fibre type can play an important role in determining meat quality characteristics such as colour. Factors such as breed, muscle type and exercise can influence the composition of the muscle with regard to fibre type and ratio. To minimise these effects, the pigs used in the present investigation were sourced from the same region, were of similar breed, housed under similar environmental conditions and slaughtered in the same abattoir.

5 MALIGNANT HYPERTHERMIA AND PORCINE STRESS SYNDROME

5.1 General

Sudden stress induced death (SDS), pale, soft and exudative (PSE) meat and halothane induced malignant hyperthermia (MH) are all manifestations of porcine stress syndrome (PSS) (Mitchell & Heffron, 1982; Louis *et al.*, 1990). The search for the basic biochemical lesion in PSS had an early breakthrough in 1966 when it was discovered that PSS was very similar to MH (Hall *et al.*, 1966).

Deaths due to MH arise from uncontrolled skeletal muscle contractions with simultaneous hypermetabolic and hyperthermic reactions. It occurs frequently at market weight (±100 kg) and after severe physical activity (Vögeli *et al.*, 1992). MH is also triggered by anaesthetics such as halothane (2-bromo-2-chloro, 1,1,1-trifluoroethane) and by depolarising muscle relaxants such as succinyl choline (Mitchell & Heffron, 1982). An impending episode of MH is usually preceded by the onset of muscle rigidity or muscle stiffness, which is indistinguishable from rigor. This occurs within 1 - 4 min of initiation of halothane anaesthesia (Harrison *et al.*, 1968) and is followed by a depletion of muscle ATP and a drop in muscle pH. Both are established causes of rigor (Szent-Györgi, 1944). Since the muscle rigidity could not be ascribed to rigor, Britt and Kalow (1970) put forward the theory that the rigidity is caused by sustained high levels of cytoplasmic Ca^{2+}.

5.2 Aetiology of MH

Pearson and Young (1989) reviewed hormonal changes associated with MH and the subsequent effect thereof on certain metabolic pathways. Epinephrine and norepinephrine are released from the adrenal medulla and stimulate glycogen breakdown in both the liver and muscles which, in turn, release glucose and lactate, respectively. In stressful situations anaerobic metabolism helps to meet the ATP demands of the muscles. This results in the formation of lactic acid, which is favoured by the action of epinephrine, and causes a build up of tissue acidity. White muscles are unable to metabolise lactic acid, and unless it is transported to the liver where it can be reconverted back into glycogen, lactic acid accretion will continue. High body (and tissue) temperatures and failure of the muscles to reduce acidity are contributing factors to the MH condition and, if left uncontrolled, can lead to death due to generalised acidosis and/or abnormally high body temperatures.

The catecholamines also stimulate fat mobilisation, yielding free fatty acids in the blood. Wood *et al.* (1977) showed that fat mobilisation can be stimulated more readily by noradrenaline in Pietrain pigs (stress prone) compared to Large White pigs. The catecholamines also accelerate heart rate and increase smooth muscle tone, thus increasing blood pressure. Hall *et al.* (1977) reported that blocking the α (but not the ß-adrenergic) pathways of the sympathetic nervous system can

successfully prevent the development of MH in Pietrain pigs. Moss (1987) established a further link between the sympathetic nervous system, catecholamines and metabolism in the PSE condition by the raised rate of utilisation of thyroxin, which is released to increase the metabolic rate, resulting in more energy being released during periods of stress.

5.3 PSS, MH and Ca^{2+} regulation in the muscle

The main sources of Ca^{2+} in the muscle are the extracellular fluids, sarcolemma, mitochondria and the SR. During MH the Ca^{2+} levels initially increase (Berman *et al.*, 1970) and since the sarcolemma is normally impermeable to extracellular Ca^{2+}, this suggests an outward movement of Ca^{2+} into the extracellular space, rather than the reverse (Britt, 1979). The sarcolemma and mitochondria each contain 10 % of the cellular Ca^{2+}, their contribution in raising the myoplasmic Ca^{2+} concentration must be small. As 80 % of all the Ca^{2+} in skeletal muscle is found in the SR (Sulakhe *et al.*, 1973), by implication the SR would be the site of the basic lesion. The Ca^{2+} release channel (ryanodine receptor) is a large protein that spans the gap between the T-tubules and the SR. This channel is activated by ATP, Ca^{2+}, caffeine, halothane and ryanodine (a plant alkaloid). Inhibition is facilitated by ruthenium red, tetracaine calmodulin, and high magnesium levels (Lai *et al.*, 1988). The Ca^{2+} release channel of the SR from MH positive pigs has a higher affinity for ryanodine binding than normal SR and therefore requires a higher concentration of Ca^{2+} to inhibit ryanodine binding (Mickelson *et al.*, 1988; Fill *et al.*, 1991).

In vivo contraction of the skeletal muscle is triggered by the release of Ca^{2+} from the SR after depolarisation of the T-tubules (Endo, 1977). Franzini-Armstrong (1970) suggested that the communication between the T-tubules and the SR occurs at the triad junction where arrays of electron dense projections, called 'foot' structures, are interposed. The use of ryanodine, which binds to the Ca^{2+} release channel in the junctional SR and modulates its activity, has allowed the purification of the ryanodine receptor (Inui *et al.*, 1987; Lai *et al.*, 1988). This purified receptor protein has been morphologically identified with the 'foot' structure (Inui *et al.*, 1987; Saito *et al.*, 1988) and has also been shown to function as a Ca^{2+} release channel when reconstituted into planar lipid bilayers (Imagawa *et al.*, 1987; Lai *et al.*, 1988). The alteration of the MH Ca^{2+} release channel was also observed in these planar lipid bilayers (Fill *et al.*, 1991).

5.4 Halotyping

With the discovery of the mutation responsible for the physiological defect associated with MH (Fujii *et al.*, 1991), a simple diagnostic tool became available for testing and clearly identifying all three genotypes associated with MH (on a large commercial scale).

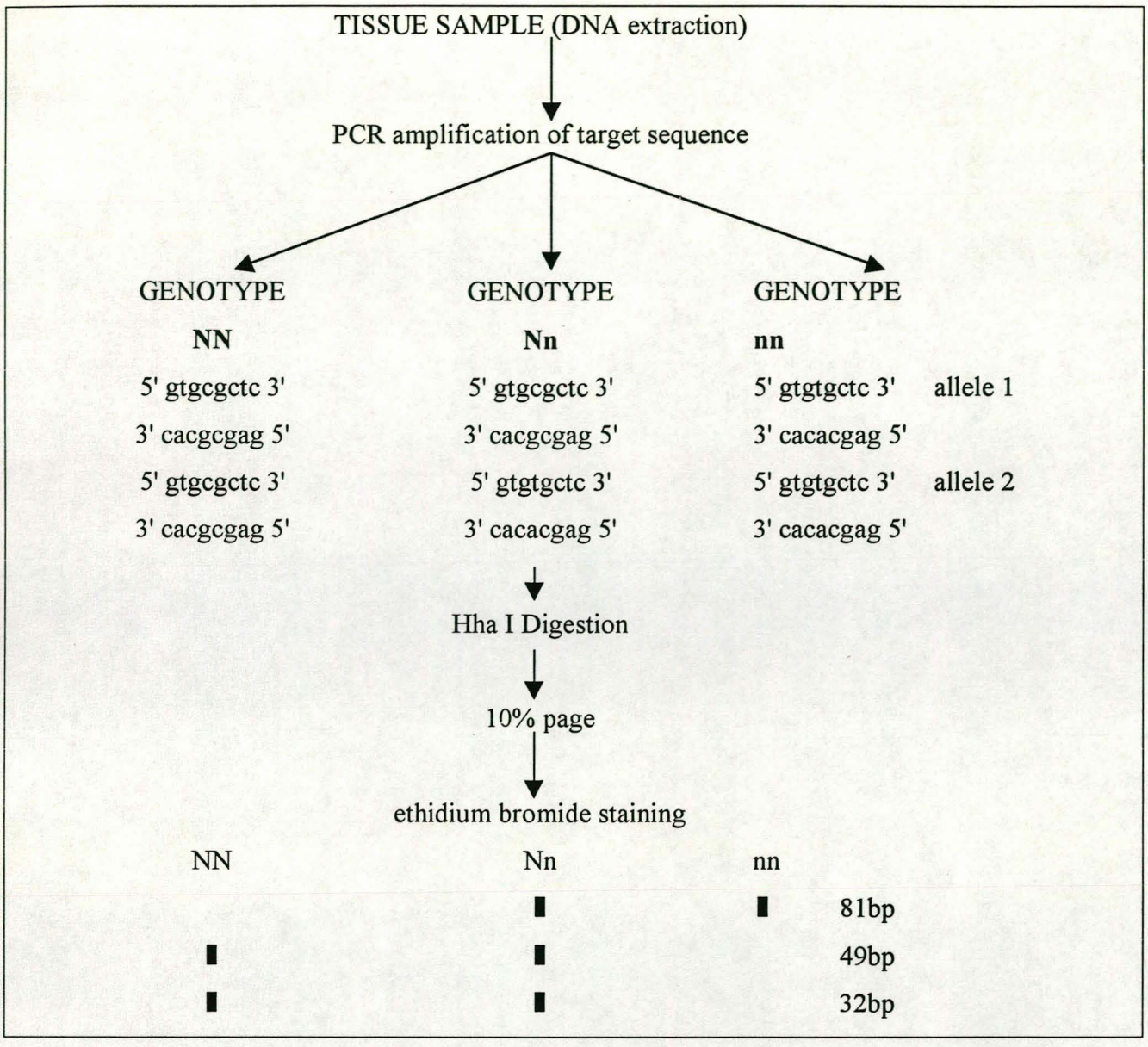

Figure 5.1.1 Diagram showing the procedure to detect the c/t transition associated with porcine MH. DNA is extracted from the tissue sample and the target region in the ryanodine receptor gene is amplified by PCR producing an 81 bp fragment. Digestion of this product by Hha I (GCG/C) yields two fragments (one 49 and one 32 bp) for normal animals (NN), three fragments (one each of 49, 32 and 81 bp) for heterozygotes (Nn) and only the 81 bp DNA fragment for mutant homozygous individuals (nn) (Houde & Pommier, 1993)

Linkage studies have traced the defect to chromosome 6 (Harbitz *et al.*, 1990) and molecular probes for a gene that codes for a calcium channel in the muscle (ryanodine receptor) has been shown to cosegregate with the halothane gene (MacLennan *et al.*, 1990). The point mutation identified by Fujii *et al.* (1991) consists of a substitution of thymidine for cytosine at position 1843 on the cDNA,

which leads to the substitution of arginine for cysteine at position 615 on the ryanodine receptor of MH carrier pigs. This transition in the DNA deletes a Hha I restriction site and creates a Hgi AI site, which forms the basis of the test, schematically represented in Fig 5.1.1. By using the polymerase chain reactions (PCR) to amplify the region where the defect occurs, it is possible to demonstrate the presence of the mutation by a restriction endonuclease digestion assay.

6 PRESLAUGHTER HANDLING, GENOTYPE AND MEAT QUALITY

6.1 General

Preslaughter handling (time of last feeding, transport, lairage, stunning, etc) affects meat quality by influencing both the rate and the extent of acidification of the muscles post mortem (Lister *et al.*, 1981). The extent of the decline in pH is determined by the energy (glycogen) concentration present in the muscle at death. A combination of high carcass temperature and a rapid rate of lactic acid formation immediately post mortem normally leads to protein denaturation and a resultant drop in WHC, as well as a pale colour, which are all typical features of PSE meat (Wismer-Pedersen, 1959a; Honikel & Kim, 1986; Offer, 1991). If preslaughter stress is more chronic, as opposed to acute stress that leads to PSE, muscle glycogen will be depleted at time of death and the decline in pH will be limited, thus leading to dark, firm and dry (DFD) meat (Warriss, 1987).

It would seem as if the interaction between the halothane genotype and preslaughter handling plays an important role in determining ultimate meat quality. Genotypes which are believed to be stress sensitive (nn) may produce PSE meat irrespective of the treatment received preslaughter, because the slaughtering procedure itself is traumatic enough to initiate its development (Briskey & Lister, 1968). The other genotypes (NN, Nn) will probably react less severely, although the presence of the halothane gene in the homozygote (nn) does seem to be involved in the severe decline of pH measured 45 min post mortem (Fisher, 1995). The importance of handling is evident from the marked differences that has been observed in the frequency of PSE carcasses between different abattoirs (Chadwick & Kempster, 1983). It is less clear whether the incidence of DFD meat is related to genotype, but there seems to be a breed effect on ultimate pH, with certain breeds less likely to develop DFD meat (Warriss & Akers, 1980). A more considerate treatment preslaughter could result in a substantial decline in the incidence of DFD meat (Nielsen, 1981).

The information available on the effects of preslaughter handling on meat quality often complicates the interpretation thereof. Many of the results come from experiments where the primary aim was not to investigate preslaughter handling. Other factors that confound such an investigation is the choice of pH values classifying meat quality (PSE, normal, DFD), halothane genotype, the muscles or muscle groups used in the assessment and the choice of stunning method. CO_2 stunning may increase the incidence of PSE to such an extent that it may overshadow any other handling effects such as last feeding, transportation and lairage (Nielsen, 1981).

Preslaughter handling can be loosely divided into four categories. The first is feeding (or withholding thereof) prior to transportation to the abattoir, the second is loading, transportation and

unloading at the abattoir, the third is the lairage period prior to slaughtering and the last is the method of stunning.

6.2 Feeding

Feed withdrawal prior to transportation and slaughtering does seem have an advantageous effect on carcasses that display inferior meat quality characteristics, such as lower carcass weight losses during chilling and cutting, lower risk of bacterial contamination of the meat during evisceration (Warriss & Brown, 1983; Lopez-Bote & Warriss, 1988), better water holding capacity (WHC) and meat colour (Warriss, 1982; Jones *et al.*, 1985; Eikelenboom *et al.*, 1991), as well as decreasing the risk of deaths occurring during transportation to the abattoir, especially during adverse (hot, humid) climatic conditions.

The time of last feeding prior to slaughtering should preferably not exceed 18 h (Warriss, 1982). The loss incurred in carcass yield due to fasting 24 h prior to slaughtering is approximately 1%, increasing to 2% or more if dehydration also occurs during transportation. Results from Jones *et al.* (1988) indicated that weight loss occurs non-preferentially from fat and lean portions of the carcass, with no difference in these losses being noted between halothane genotypes. Experiments done by Nielsen *et al.* (1979) on the effect of time of last feeding and different holding periods (lairage), indicated that pigs that have been fed prior to slaughtering, combined with a short holding period, gave higher frequencies of PSE meat, compared to pigs with no feeding and a prolonged holding period. However, the frequency of DFD meat was higher in the unfed pigs. In addition, the DFD frequency increased during the first couple of hours of the holding period. By manipulating feeding and holding period one could therefore minimise the frequency of both DFD and PSE meat. This can be achieved by slaughtering unfed pigs immediately after unloading, and slaughtering fed pigs after approximately four hours of lairage. Thus, regulating the frequency of deviating meat quality (PSE, DFD) is possible, but implementation of the above mentioned could prove difficult in practice, since it may differ from farm to farm. In the present investigation food was withheld for approximately 10 hours to minimise stress during transportation and at the time of slaughtering

6.3 Transportation and lairage

There seems to be general agreement amongst researchers that transportation, especially loading and unloading, is a potentially stressful experience for pigs. The interaction of the halothane genotype with handling can produce a wide variety of results, with the stress-susceptible pigs producing a higher frequency of PSE meat after short transportation distances (Warriss, 1987).

Stress-resistant pigs will generally develop PSE only after severe but short term stress, or DFD after prolonged stress (Barton-Gade *et al.*, 1988).

During transportation the pigs are exposed to various stressors such as noise, vibration and temperature extremes. These stressors lead to certain metabolic and physiological changes within the animal. During exposure to simulated loading, transportation and lairage, researchers found that hormone levels, such as cortisol and thyroxin, increased rapidly and for increased periods of time, suggesting high levels of stress being suffered by the pigs (Moss, 1984; Spencer *et al.*, 1984). The dehydration seen in transported pigs could be associated with these hormonal changes (Warriss & Brown, 1983). Other physiological changes associated with this stress period are increased heart beat (Augustini & Fischer, 1982) and body temperature (Augustini & Fischer, 1982), both increasing with higher stocking density. This increase in body temperature will be reflected as a higher post mortem muscle temperature, thus creating ideal conditions (together with a rapid drop in pH) for the development of PSE meat (van der Wal & Eikelenboom, 1984).

It would seem as if the interaction of genotype with handling (transportation, loading, unloading) is of a complex nature. Results from Schworer *et al.* (1981) indicate that, for stress resistant pigs, a short transportation time combined with other stress inducing handling practices preslaughter, does not seem to affect meat quality. However, the effect on meat quality is quite detrimental in the case of stress susceptible pigs. Measuring creatine phosphokinase levels, as an indicator of stress susceptibility, Barton-Gade (1984) reported that stress susceptible (nn) genotypes gave poor meat quality irrespective of preslaughter handling. Transportation experiments also gave conflicting results. Cuthbertson and Pomeroy (1970) found no significant differences in meat quality when comparing pigs transported for 0.5 or 8 h preslaughter. In contrast, Lendfers (1968) reported that transportation of pigs over long distances (200 - 300 km) led to a higher incidence of PSE than short (20 - 80 km) journeys. In Danish Landrace pigs, longer transportation times (0.25 h up to 3 h) reduced the incidence of PSE and slightly increased the incidence of DFD meat (Buchter, 1974). Barton (1974) confirmed these results by experimenting with different transport conditions. The incidence of PSE was highest with a transportation time of 0.5 h and lowest with 1.3 h, with a pronounced genotype interaction. The ultimate pH was also found to be higher in several red muscles. The effect of prolonged transportation times can be quite dramatic (Barton, 1974). Pigs transported in well equipped vehicles with adequate ventilation, partitions and non slip floors resulted in a decrease of 25% in PSE frequency when transportation time was increased from 15 min to 3 h. There was a corresponding increase in the frequency of DFD meat. Augustini and Fischer (1981) reported that a long period of transportation and/or lairage can result in an increase

of both PSE and DFD meat in stress susceptible pigs, as some of them may relax and recoup energy losses.

Lendfers (1968) reported that meat quality decreased in pigs transported at higher stocking densities (0.35 m^2/pig) and poor ventilation, compared to those transported in vehicles with good ventilation and lower stocking densities (0.66 m^2/pig). In practice, recommended stocking densities are rarely adhered to and overcrowding is frequently encountered (Guise & Penny, 1989). This can cause fighting which results in skin damage and a high incidence of prolapsed rectum. Floor area allowed should be 0.5 - 0.6 m^2/pig for pigs of 90 kg live weight and slightly more for heavier pigs (Barton-Gade *et al.*, 1988). Factors affecting body temperature at slaughter are of interest because of the contribution of muscle temperature in the development of PSE. Higher ambient temperatures (25°C) have been shown to be associated with an increase in meat quality deterioration in Dutch slaughter pigs (Lendfers, 1968). Barton (1971) also observed a significant seasonal effect in Danish pigs. The poor meat quality in summer and autumn was largely due to an increase in the extent of deviation from normal meat, and not to more pigs having meat quality deviating from the normal. Long and Tarrant (1990), using pigs from a single source, found no differences in body temperatures or MLT temperatures between pigs slaughtered in summer (mean temp. = 19.5°C) or winter (mean temp. = 11°C). Using a wider range of temperatures (12° to 24°C), Warriss (1991) found a positive correlation between ambient temperature, MLT muscle temperature at 1 h post mortem and drip loss. It is, however, unclear whether the observed effects were due to the influence of high ambient temperatures or the cooling rate of the carcass on the killing line. Long and Tarrant (1990) found that cold water showering prior to slaughtering brought about a drop in loin muscle temperature, which was sufficient to reduce paleness and drip loss in loin chops. There is general agreement that transport is a potentially stressful experience for pigs. Pigs with the halothane gene, particularly the halothane positive (nn) pigs, can have a very pronounced reaction, with increased incidences of PSE meat after short transportation distances. Improvements in the transportation conditions can lead to improved meat quality, as well as a decrease in the incidence of deaths during transit and is also in the interest of better animal welfare (Hails, 1978).

Holding in lairage may have negligible or no influence on meat quality, particularly in pigs that have not been subjected to stress caused by factors such as handling and transportation, or are resistant to stress (Warriss, 1987). Stein (1978) compared lairage for 0, 8 and 24 h. The lowest incidence of PSE was after a lairage period of 8 h, and the highest after no lairage. The results indicated that a lairage time of not less than 6 h is recommended. Augustini and Fischer (1981) found that lairage times up to 6 h decreased the incidence of carcasses with low initial pH values.

Longer holding times resulted in a slight increase in the incidence of DFD carcasses. It was found that overnight lairage caused an increase in the incidence of DFD carcasses, as well as a slight increase in PSE, the latter being difficult to explain. Research by Barton (1971) illustrated the different effects observed in different metabolic muscle types. Short periods of stress promoted the development of PSE, particularly in the MLT, but to a lesser extent in the *M. biceps femoris.* The MLT of pigs is a glycolytic (white) muscle, whereas the *M. biceps femoris* contains more myoglobin and is classified as an oxidative or red muscle (Beecher *et al.*, 1965a, b; Bendall, 1975). The incidence of PSE was reduced with longer periods of stress, but this was associated with an increase in the incidence of DFD. The author concluded that lairage of 1h to 2 h did not necessarily improve meat quality.

It seems that lairage time does not have much influence on meat quality in stress resistant pigs, unless prolonged when an increase in DFD carcasses may occur. For stress susceptible or stressed pigs a short lairage period may allow for some recovery from previous handling or transportation stress and thus reduce the incidence of PSE carcasses. The best results seem to be if lairage time is in the order of approximately 6 h. Extension of this time can lead to DFD meat, particularly in pigs fatigued by long transportation or feed withdrawal. By reducing muscle glycogen stores the potential development of PSE carcasses is lessened. However, it is unclear whether this or the lairage period is more important in reducing the incidence of meat with a low initial pH. The pigs in the present investigation were slaughtered after a 2 h lairage period to simulate current abattoir practices, and may have allowed for partial recovery from the short transportation prior to slaughtering.

6.4 Stunning

The principal stunning methods currently used by abattoirs are electrical or carbon dioxide stunning, but captive bolt, percussion bolt and free bullet methods are used on occasion (Gregory, 1987). Use of the captive bolt has certain disadvantages, such as insufficient bleeding of the carcass, which leads to spoilage, increased incidence of blood splash, violent convulsions after stunning and the high incidence of PSE meat. During the 1930's electrical stunning surpassed the use of captive bolt, since it is easier to administer and makes handling of the animal easier (Muller, 1932). Warrington (1974), Marple (1977) and Warriss (1977, 1984) have reviewed carcass and meat quality defects associated with stunning and slaughter. These defects included bone fractures, blood splash, bruising, inadequate bleeding and PSE carcasses.

The PSE condition of the carcass is exacerbated by excessive stimulation or stress prior to sticking, thus necessitating the proper handling of the animals to minimise stress and muscular activity in all stunning and slaughtering methods (Gregory, 1987). With stunning, the normal metabolical processes are severely disrupted, thus contributing to post mortem meat quality deviations. During stunning, the adrenal medulla excretes a large amount of catecholamines, which activate muscle glycogenolysis and thereby increasing the availability of substrates for acid (lactate) formation. This increase in catecholamines can be observed immediately after stunning, irrespective of the stunning method used (Van der Wal, 1975). One of the consequences of stunning is muscular contractions (Van der Wal, 1978a), which, together with the high levels of catecholamines, lead to significantly increased levels of lactate. Pigs stunned with carbon dioxide were thought to be particularly prone to the formation of PSE carcasses because of the stress associated with the process, but the use of the Compact stunner, which operates along the lines of a Ferris wheel, decreased the amount of PSE carcasses to below that of electrical stunning (Larsen, 1982; Barton-Gade, 1984). The effect of electrical stunning techniques on meat quality have been widely documented (Overstreet *et al.*, 1975; Van der Wal, 1978b; Schutt-Abraham *et al.*, 1983; Rothfuss *et al.*, 1984). The results indicate that excessive stunning can lead to the development of PSE meat, regardless of whether they were positive (nn) or negative (NN, Nn) halothane reactors. The stunning used in this investigation was a 250V AC current applied ear to ear for 3 – 5 seconds, which seems a good compromise between the voltage and stunning time used, and strict control was exercised to ensure that no overstunning or excessive stunning took place, which could exacerbate the PSE condition, especially for the nn pigs.

In conclusion from the foregoing literature review, it is clear that handling technique may influence different genotypes differently. On commercial farms producing a mixture of genotypes, it will become extremely difficult to manage different handling techniques for different genotypes, since, in most cases, the genotype will not be known.

7 CARCASS AND MEAT QUALITY CHARACTERISTICS

7.1 General

Various studies seem to indicate that the presence of the halothane gene is accompanied by decreased meat quality characteristics, in particular a higher incidence of PSE meat (Lundström *et al.*, 1989; Murray *et al.*, 1989; Sather *et al.*, 1991a, b; De Smet *et al.*, 1992; Leach *et al.*, 1996; Fisher & Mellett, 1997). However, producers and breeders have remained interested in the use of the gene, since it is associated with an increase in carcass lean contents, higher growth rates and improved food conversion ratios (Aalhus *et al.*, 1991; Pommier *et al.*, 1992). Halothane positive (nn) genotypes are generally assumed to yield more lean meat when compared to halothane carrier (Nn) and negative (NN) genotypes, but with an adverse effect on meat quality (rate of pH decline, colour, WHC) and a higher incidence of PSE (Jones *et al.* 1988).

7.1.1 Midline measurements

Comparison of backfat thickness between the three halothane genotypes have resulted in various and often conflicting results. Jones *et al.* (1988) compared backfat depths of the three genotypes (NN, Nn and nn) measured at different locations (shoulder, mid back, back, loin, lumbar) and for the majority of the comparisons the backfat thickness between nn and NN genotypes were not significantly different. Of interest is that the fat depth of the nn genotypes at the loin area was significantly more ($P < 0.05$) than that of the NN genotypes. The Nn genotypes tended to have more backfat than either the NN or nn genotypes, although few of the comparisons were statistically significant. Comparison of NN and Nn genotypes (Leach *et al.*, 1996) also failed to show any significant differences in midline fat measurements between genotypes.

Generally, dorsal midline fat measurements on split carcasses in the shoulder region have a low predictive value for lean content, whereas the lumbar and posterior thoracic region show the highest predictive value (Kempster & Evans, 1979; Fortin *et al.*, 1981). The reasons for this are not clear; it is possible that the shoulder measurements, which are furthest from the initial splitting point, is more sensitive to the accuracy with which carcasses are separated when hanging by the hind legs. Another source of inaccuracy is that the shoulder fat is less well supported by the vertebral spines than the rump fat. Also, the *M. gluteus medius* provides a clear anatomical point of reference for midline measurements (Kempster *et al.*, 1982).

Results from various studies (Eikelenboom *et al.*, 1980a; Andresen *et al.*, 1981; Jones *et al.*, 1988; Simpson & Webb, 1989) showed that the nn genotypes have shorter carcasses than NN genotypes, with Nn genotypes showing an intermediate position. Results from Pommier *et al.* (1992) showed

no significant differences in carcass length between NN and Nn genotypes. However, it must be kept in mind that the live weights in the above mentioned studies ranged from approximately 85 kg to over 110 kg, which could influence gene expression with regard to certain carcass traits.

7.1.2 Lateral measurements

Carcass measurements taken laterally to the dorsal midline over the MLT appear to be more precise predictors of carcass lean compared to measurements taken at the dorsal midline (Fortin *et al.*, 1984). After investigating the relative precision of lateral fat thickness measurements along the carcass, Kempster and Evans (1979) concluded that the area over the MLT in the region of the last and third/fourth last rib had the highest predictive value for assessing carcass lean yield. Results from Fortin *et al.* (1984) indicated that fat thickness measurement was the major component of the prediction of percentage lean yield, and that the addition of a muscle depth measurement taken at the same location, although significantly reducing the residual standard error, only caused a minor improvement in precision for predicting lean yield.

The percentage predicted lean meat in a pig carcass in South Africa is based on a single measurement with a Hennessey Grading Probe (HGP) or an Intrascope, on a position between the 2nd and 3rd last ribs, 45 mm from the midline, while the carcass is in a hanging position (Government Notice No. R. 1748, 26 June 1992). The percentage meat (LMP) is calculated, depending on the technique, by means of the following formulae:

HGP: $LMP = 72.5114-(0.4618 \times FT) + (0.0547 \times MD)$

Intrascope: $LMP = 74.4367 - (0.4023 \times FT)$

Examining the formula for predicting percentage lean meat (with HGP) in the carcass, it is clear that the fat thickness (FT) on the measurement position makes a much larger (and negative) contribution (approx. 10 times) to predicting lean meat yield than the muscle depth (MD) on the same position.

Various studies reported that the Nn genotype had a fat thickness intermediate to the homozygotes (NN, nn), with NN having the highest and nn the lowest values (Eikelenboom *et al.*, 1980a, b; De Smet *et al.*, 1992; Fisher *et al.*, 1994). Sather *et al.* (1991a), comparing NN and Nn genotypes, reported no significant differences in backfat thickness, although the Nn genotypes tended to have a lower value. Pommier *et al.* (1992) reported similar results. Jones *et al.* (1988) indicated that, although not significant, Nn genotypes tended to have higher backfat values than both NN and nn

genotypes. Comparison of gender (barrows vs. gilts) in studies (Jones *et al.*, 1988; Sather *et al.*, 1991a; Pommier *et al.*, 1992) consistently showed that gilts have significantly lower backfat thickness values at the measuring site than barrows, and thus a greater percentage predicted lean meat yield at similar carcass weights, regardless of halothane genotype. Jones *et al.* (1988) reported that although the nn and NN carcasses had similar fat thickness measurements, the nn genotypes had a lower proportion of carcass fat compared to the NN genotypes. Breed may also play an important role, as a comparison of NN and nn genotypes from both the British Landrace and Pietrain/Hampshire lines indicated that the advantage in lean yield for nn genotypes was much smaller in the British Landrace compared to the Pietrain/Hampshire, suggesting that the result was possibly caused by different genetic backrounds resulting in less extreme muscle development in the former (Webb & Simpson, 1986). Researchers (Jones *et al.*, 1988; Sather *et al.*, 1991a; Pommier *et al.*, 1992) do emphasise that a single measurement of fat thickness may not necessarily identify the greater lean meat content of genotypes with the halothane gene in homozygous (nn) or heterozygous (Nn) form.

Comparison of MLT width and depth showed that the nn genotypes have larger loin eye muscles than the NN and Nn genotypes (Jones *et al.*, 1988). In concurrence, Fisher *et al.* (1994) reported similar results for MLT depth, however, differences observed between the three genotypes were not significant. Results from Nel *et al.* (1993), comparing NN genotypes to pooled Nn and nn genotypes, indicated that MLT muscle depth at the same point of measurement differed significantly between the two groups, with the pooled Nn and nn group having a higher value. Similar differences between NN and Nn genotypes were reported in various investigations (Sather *et al.*, 1991a; Pommier *et al.*, 1992; Leach *et al.*, 1996), although a few of the differences were significant. Comparison of all three genotypes suggest that the nn genotypes have larger MLT muscles, but similar backfat measurements compared with the other two genotypes, whereas the Nn genotypes tend to be intermediate for MLT measurements (Jones *et al.*, 1988; Simpson & Webb, 1989).

7.1.3 Cut distribution and carcass composition

A number of the investigations conducted have found that the Nn genotypes tend to be intermediate for carcass lean yield compared to the NN and nn genotypes (Webb *et al.*, 1982). Similarly, other investigators (Andresen *et al.*, 1981; Sather & Murray, 1989; Sather *et al.*, 1989) reported carcass lean yield from Nn genotypes to be approximately equal to the mid value of their parental lines (NN and nn) at final farm weights (90 - 100 kg). However, some studies have found seemingly conflicting evidence.

Jones *et al.* (1988) reported that nn carcasses had a significantly higher proportion of lean and a lower proportion of bone, skin and fat compared to the NN carcasses. The Nn carcasses had more lean, less skin and bone and similar fat content to the NN carcasses. Sather *et al.* (1989) reported that predicted lean yield did not differ among genotypes. Similarly, Sather *et al.* (1991a) and Pommier *et al.* (1992) found no significant differences in lean meat content comparing NN to Nn genotypes. It should also be noted that carcass evaluation techniques vary widely among different countries. Another source of variation is the method used to express carcass composition, e.g. lean content calculated as a proportion of the primal weight of the four primal cuts (picnic, butt, loin, ham) or as a proportion of the whole carcass. This could add to inflated differences between genotypes and sexes.

Composition of the four primal cuts show that the nn genotypes consistently have more lean than NN genotypes (Jones *et al.*, 1988). Bone, skin and fat from the carcasses of nn genotypes also had lower proportions (expressed as a proportion of the total weight of the primal cuts) than the cuts from the NN genotypes. Lean content of the loin, ham and shoulder were not affected by genotype in a comparison of NN and Nn genotypes (Pommier *et al.*, 1992). In a similar investigation, Leach *et al.* (1996) found that lean yield was significantly higher for carriers (Nn) compared to NN genotypes. Some studies (Sather *et al.*, 1991a) suggest a genotype by weight interaction, with the relative differences in carcass composition between genotypes changing with an increase in slaughter weight. However, Leach *et al.* (1996) did not report any interaction suggesting a linear increase in fat free lean content with an increase in slaughter weight. In this investigation a fixed slaughter weight (± 86 kg) was used for all three genotypes and both sexes, which is considered the ideal weight of baconer pigs by the South African pork industry. Lean yields were expressed as a percentage of the primal cuts, as well as a percentage of cold carcass weight to compensate for some of these interferences. Full statistical models were also fitted to test for sex interaction with genotype at a fixed live weight (See: # 10.10 Statistical analyses).

7.2 Meat quality

Ultimately meat quality is determined by both genetic and slaughter conditions. The development of PSE meat is greatly affected by pre-slaughter handling and slaughter methods (Tarrant, 1989). However, there is still some question about which factors (genetic or environmental) are the most important in influencing meat quality.

7.2.1 Initial and ultimate pH

Comparison of the three genotypes (Murray *et al.*, 1989; De Smet *et al.*, 1992; Fisher *et al.*, 1994) has shown that the halothane gene is associated with both a rapid decrease in pH as well as a low initial pH (pH_i). This, combined with high initial carcass temperatures, contribute significantly to protein denaturation (Wismer - Pedersen, 1959a, b; Offer, 1991), resulting in the pale colour and reduced WHC typical of PSE meat. Comparison of PSE (pH_{45} < 5.8), normal and DFD MLT (ultimate pH > 5.87), with regard to the post mortem pH decline rate, indicated significant differences between PSE and DFD at pH_i, as well as between PSE and normal meat. The development of PSE characteristics in the muscles thus seems to be initiated by a combination of a lower muscle pH already at exsanguination due to lactate accumulation before slaughter, and a faster pH decline post mortem. The rate of pH decline is determined by both time post mortem and the actual initial pH level (Bendall *et al.*, 1963).

Under a constant temperature of 37°C, Bendall *et al.* (1963) reported that normal muscles had a biphasic decline in pH_i with a lower initial rate of decline when pH_i > 6.5 (90 min post mortem). In contrast, PSE muscles had a constant rate of pH decline during the first 150 min post mortem. Enfält *et al.* (1993) also reported a linear decline in pH for PSE muscles, as opposed to the biphasic pattern for normal muscles. Results from De Smet *et al.* (1992) indicate a sharp decline in pH_i as lean contents increase in nn genotypes, with only a small reduction for NN and Nn genotypes. The mean initial pH of the NN and Nn genotypes remained well above 5.8, even in the highest lean content class, suggesting that a slight depression in pH of NN and Nn genotypes does not necessarily mean an increased risk of PSE under proper slaughter conditions.

Evidence regarding ultimate pH (pH_u) indicate no significant differences in pH_{24} between the three genotypes (Murray *et al.*, 1989; Fisher *et al.*, 1994). Comparisons of pH_u values between NN and Nn genotypes is also less clear cut. Many studies (Sather *et al.*, 1991a; Pommier *et al.*, 1992; NPPC, 1994) reported insignificant differences between NN and Nn in pH_u measured in the MLT. Sather *et al.* (1991b) reported a halothane genotype by slaughter weight interaction for meat quality; Nn genotypes had meat quality (pH) similar to NN genotypes at lighter carcass weights but more were comparable to nn genotypes at heavier carcass weights. Studies by Leach *et al.* (1996) and Sutton *et al.* (1997) found no evidence of a weight interaction, suggesting that meat quality was maintained across the weight ranges investigated. Cisneros *et al.* (1996) reported no effect of slaughter weight at pH_{45}, but observed a linear decrease in pH_u as slaughter weight increased above 140 kg live weight.

Using biopsy samples of crossbred (Landrace x Large white) Nn genotypes, Cheah *et al.* (1995) showed that innate variations in meat quality, as defined by pH_1, do exist, with 43 % of the pigs classified as normal and 57 % of the pigs classified as PSE. However, Kauffman *et al.* (1993) concluded that ultimate pork quality can only be assessed reliably after the development of full rigor. They emphasize that pH_{45} can be used successfully when groups of carcasses, rather than single carcasses, are screened for quality variations when conducting pork quality research. In the present investigation, both pH_{45} and pH_{24} were recorded and analysed.

7.2.2 Colour

Meat has a number of colour components such as the dark red colour of myoglobin, the bright red colour of oxymyoglobin and the brown colour of metmyoglobin. Another colour component is the green pigment sulphmyoglobin (formed by bacterial activity), although rarely seen in the normal course of meat handling (Swatland, 1994). There are several different approaches to meat colour measurement: absorbance spectrophotometry, reflectance spectrophotometry and subjective evaluation. However, the choice of which system to use depends largely on what equipment is available. In the method recommended by the International Commision on Illumination (CIE), the primary hues, red, green and blue, are added and subtracted from each other to match any colour. The 1976 CIE L* (brightness), a* (red-green range), b* (blue-yellow range) colour space, known as CIELAB, is a non-linear cube root transformation of the 1931 tristimulus values to approximate the 1931 Munsell system. CIELAB is the colour space most used for general application (MacDougall, 1988) and it has been proposed that all results (regarding meat science) should be reported using the CIE system (Swatland, 1994).

Comparison of colour (CIELAB) and reflectance (GÖFO) values showed that the presence of the halothane gene is accompanied by an increase in reflectance and a paler colour (Murray *et al.*, 1989; De Smet *et al.*, 1992; Leach *et al.*, 1996), which is partly due to denaturation of the sarcoplasmic proteins. Various factors, such as chilling rate, rate of pH decline and available substrate (glycogen) for conversion to lactic acid contribute to the variation in opacity of raw meat. Meat colour is not constant along the MLT, with the cranial part of the muscle being less susceptible to aberrant meat quality than the lumbar region (Van der Wal *et al.*, 1987). However, as noted by Swatland (1995), low pH does not necessarily cause meat paleness, nor does a high pH cause darkness. The hypotheses of Bendall and Wismer-Pedersen (1962) that light scattering at a low pH could be due to protein denaturation, and Hamm (1960) that the shrinkage of myofibrils at a low pH increases the difference in refractive index between the myofibrils and sarcoplasm, contribute to an explanation of light scattering. However, only the hypothesis that light scattering is due to myofibrillar

birefringence is supported by experimental data, indicating that a decrease in pH is accompanied by a decrease in birefringence, that is that less light is transmitted through the fibre and more light is scattered compared to a fibre with a high pH and low birefringence. This lack of a comprehensive theory with accompanying data highlights the difficulty in assessing pork meat quality based on either colour or pH measurements alone. In the present investigation the CIELAB system (L*, a* and b* values) was used to evaluate meat colour, with L* indicating brightness, a* the red–green range of the colour spectrum and b* the blue-yellow colour range.

7.2.3 Drip loss

Various studies indicate that the presence of the halothane gene is accompanied by an increase in drip loss, when compared to meat from pigs that do not possess the halothane gene (Lundström *et al.*, 1989; Murray *et al.*, 1989; Sather *et al.*, 1991a, b; De Smet *et al.*, 1992). Comparison of the three genotypes (NN, Nn and nn), with regard to drip loss from the MLT muscle show that the nn genotype have higher percentage drip losses than the NN genotype (Murray *et al.*, 1989). Likewise, comparison of NN and Nn genotypes indicated that the carcasses from Nn genotypes tend to have a higher drip loss than NN genotypes (De Smet *et al.*, 1992; Leach *et al.*, 1996; Sutton *et al.*, 1997). Sather *et al.* (1991a) reported that a significant increase in drip loss was observed at both the anterior end and at the centre of the MLT derived from Nn genotypes when compared to NN genotypes. However, this was one quarter to one half less than the differences previously reported by Murray *et al.* (1989). Lundström *et al.* (1989) reported that drip loss from Nn genotypes were intermediate to the homozygous (NN, nn) genotypes, but Murray *et al.* (1989) found that the drip loss from the Nn and nn genotypes were similar. In the present investigation portions of the MLT, with adhering fat and bone, from the thoracic region (6^{th} thoracic vertebra) was used to determine drip loss.

7.2.4 Chemical composition and eating quality

Results from Murray *et al.* (1989) showed that the presence of the halothane gene is accompanied by a significant decrease in intramuscular fat and increase in protein content, with the nn genotypes having meat with the highest protein and lowest fat contents, and NN genotypes the lowest protein and highest fat values. Similar to carcass lean and fat composition, the intramuscular fat and protein composition of Nn genotypes tended to be close to or identical to the NN genotypes.

Similarly, Warner-Bratzler shear values indicated that the meat from nn genotypes had the highest values, with Nn intermediate and NN the lowest. Environmental influences, such as stress before slaughter (due to the prolonged use of electric goads), induces glycogen depletion, high ultimate pH

and ultimately DFD meat, and also causes favourable changes in cooked meat aroma, flavour, texture, tenderness and juiciness (Swatland, 1994). In beef, DFD occurs regularly in young bulls, particularly in the MLT, *M. semitendinosus*, *M. semimembranosus* and *M. gluteus medius* muscles, whereas in other muscles, pH values may be near to normal (Tarrant & Sherington, 1980). Taste panel evaluation have indicated that cuts from DFD meat are similar to normal beef for softness, juiciness and tenderness (Swatland, 1995). There is controversy in the literature concerning the tenderness for PSE meat, compared to normal meat. Some authors reported a higher tenderness for PSE pork (Kemp *et al.*, 1976), whereas others found normal meat more tender (Buchter & Zeuthen, 1971). Tornberg *et al.* (1992) concluded that, as tenderness is best correlated to the sarcomere length of raw meat, tender pork could be obtained if the meat has a low degree of contraction when rigor mortis sets in. In the present investigation, proximate chemical analyses (protein, moisture, fat and ash) of fresh samples from each genotype was carried out according to the standard AOAC techniques (AOAC, 1984).

7.3 Conclusion

In conclusion, pork quality is the result of breeding, management systems, preslaughter handling practice, slaughter hygiene, product technology and product distribution. Although many recommendations can be made at present to reduce the incidence of PSE pork, it remains unclear which factors are the most important in influencing meat quality. It is therefore in the interest of the pork industry to keep abreast with the latest technology and information available, and in so doing they will be able to manipulate meat quality to the advantage of producers and satisfaction of consumers.

8 PROCESSED PORK PRODUCTS

8.1 General

Inferior pork products or product failures due to changes (physical, biochemical) in raw pork are mainly due to pH related properties such as PSE, DFD and low ultimate pH (Møller *et al.*, 1992). The association of the halothane gene with inferior fresh meat quality, especially in the nn genotypes, is well documented and is therefore of interest to both producers and processors, since it determines to a large extent certain quality characteristics of value added meat products. The body of literature available on processed pork is vast, but little information is available on the effect of the halothane gene on processed product quality. The present investigation is therefore concerned with the effect of all three genotypes on carcass characteristics, meat quality characteristics, selected processing characteristics of a number of products, as well as to determine whether certain processing additives can alter some of the deleterious effects associated with PSE meat during processing.

8.2 Cured products

Cured products, such as pork bellies and back bacon, made from PSE meat have a lower net gain compared to normal (non-PSE) meat (Wismer-Pedersen, 1968). Fisher and Mellett (1997) reported a significantly higher ($P < 0.001$) moisture loss for the nn genotypes during curing of back bacon, resulting in a lower final yield. When curing PSE meat (with known reduced WHC) the net gain during curing is less than that of normal meat. However, curing whole sides with intact membranes reduces the amount of fluid loss (Wismer-Pedersen, 1968). The reduced WHC of PSE meat leads to an increased percentage gelatinous cookout (% jelly) in the canned hams due to a higher than normal degree of aggregation of meat proteins, especially when pasteurized (Wismer-Pedersen, 1968). Hams from PSE meat, processed with polyphosphates, showed significant improvement in WHC and technological yield (Davis *et al.*, 1975a, b). Honkavaara (1988) compared PSE pork ($pH_1 < 5.8$) with non-PSE pork ($5.8 < pH_1 < 6.4$) in cooked cured ham production. PSE pork resulted in an acceptable product, yet with decreased sensory scores and a technological yield of 94.0 % compared to 105.9 % for non PSE ham. Similar relationships between pH and ham quality was reported by Müller (1991). Increased pH values resulted in higher cooked ham yields, with a concomitant decrease in the amount of juice exudation. The hams with the higher pH levels were also more tender.

Comparison of conventional with hot processing in terms of yields and major quality traits of bacon indicated that neither of the two processes showed any significant advantages. Neel *et al.* (1988) observed no significant change in post mortem treatments such as smokehouse yields or slicing

yields. Sensory attributes appear unaffected by time of processing, except for crispness ratings, which is lower for hot processed bacon (Abu-Baker *et al.*, 1983; Neel *et al.*, 1988). This investigation measured and recorded initial gains and final yields on back bacon on all three genotypes, as well as determining the percentage cooking loss on canned hams, manufactured from the pooled meat from each genotype.

8.3 Meat quality and the acid/base status of the pig

A report on treatment of halothane positive (nn) genotypes with ammonium chloride, sodium bicarbonate, and water (control) in an effort to improve the acid/base balance and thus meat quality, suggested that oral loading of nn genotypes with these chemicals were not wholly effective in preventing PSE development, and that the strong homeostatic mechanisms involved in maintaining acid/base homeostasis in the living animal countered the effect of the alkaline salts (Shand *et al.*, 1995). No such treatment was thus applied in this investigation.

8.4 The role of salt and various phosphates on product quality

Various additives are used in the curing process, and, although the focus of this investigation was primarily on the effect of the halothane gene, some of the adjuncts used in meat processing have a direct bearing on product quality in relation to the quality of meat used for manufacturing.

Meat contains both salt soluble and water soluble proteins (Ellinger, 1972). Saffle (1968) defined salt soluble proteins (e.g. actomyosin) as those proteins that are soluble in solutions of NaCl, and insoluble in water. Water soluble proteins, such as the sarcoplasmic proteins, are soluble in pure water and insoluble in salt water. The importance of the salt soluble proteins is their ability to emulsify fat, and together with certain phosphates, cause the dissociation of the actomyosin complex (Ellinger, 1972), which is necessary for increased water retention and meat binding during coagulation or heat treatment.

Water uptake during curing appears to be due to lateral expansion of the myofibrils, accompanied by protein denaturation (Varnam & Sutherland, 1995). A number of hypothesis explaining this phenomenon exist, amongst these the theory that the mechanism of swelling is due to an increase in the negative charges on the filaments due to the binding of Cl^- ions. This in turn increases the repulsive electrostatic forces, and thus swelling takes place. Divalent cations such as Ca^{2+} and Mg^{2+} lower the WHC of meat by reducing the electrostatic repulsion between negatively charged groups. This tightens the muscle structure, causing shrinkage (Pearson & Young, 1989). Magnesium salts have a much stronger effect on WHC than sodium salts of the same ionic strength, because of the

stronger bonding between magnesium and the myofibrillar proteins. With the use of NaCl and $MgCl_2$, shrinkage does not occur because the chlorine ions superimpose the effect of cations on WHC. The WHC of meat is also increased by the removal of the divalent cations by exchange resins or other similar methods (Pearson & Young, 1989). In the present investigation NaCl was used to enhance WHC to its maximum.

8.4.1 Phosphates

The retention of water in cured meat products is very important to their palatability, both for comminuted and whole muscle products. Numerous reviews discuss this particular aspect of the food phosphates, such as the effect of phosphate additives effect on moisture retention through their ability to solubilize meat proteins (Swift & Ellis, 1956).

In a review by Hamm (1971) on the interaction of phosphates and meat proteins, it was pointed out that the basic phosphates (e.g. sodium tripolyphosphate) exert a stronger hydrating effect on meat than the neutral (tetrasodium pyrophosphate) or acidic phosphates (sodium acid phosphate). The role that phosphates play in meat hydration is not fully understood, although several mechanism have been suggested. These include the changes associated with pH value, effects on ionic strength and the specific interactions of phosphate anions with divalent cations and myofibrillar proteins. A major function of phosphates seems to be the loosening of the tissue structure due to complexes formed with calcium. Other probable consequences of phosphate/protein binding and cleavage of actomyosin are the swelling of the muscle fibre system and thus increased water uptake. The increased ionic strength may also lead to reduced interaction between proteins and the formation of a colloidal solution of myofibrillar proteins (Varnam & Sutherland, 1995).

Grau (1958), discussing the role of phosphates and water binding in meat products, indicated that the addition of phosphates does not increase the water absorption of meat, but rather caused meat products to bind water more completely and in so doing prevent losses during processing. This probably explains the results from various researchers (Wasserman, 1957) that cured meat products with added phosphate, including sausage products, bind more water, are plumper when encased in sausage casings and lose less water during smoking and heat processing. The addition of 0.35% polyphosphates to canned hams reduced the free juices by 5% and improved their firmness (Siedlecki, 1965, as cited by Ellinger, 1972).

The effect of pH on aspects such as protein extraction and water binding have been thoroughly studied (Fukazawa *et al.*, 1961a,b; Saffle, 1968). Manipulation of meat pH to values well below 5.5

(iso-electric point of meat proteins) indicated that pH values of less than 4.5 were characterized by increased water absorption and binding. pH values of approximately 7.0 was associated with increased protein extraction, especially in the presence of phosphates, with pyrophosphate being more effective than tripolyphosphate and hexametaphosphate (Fukazawa *et al.*, 1961b). Controls containing NaCl also showed increased amounts of extracted proteins with an increase in pH, but to a lesser extent than in the presence of phosphates. Results from Davis *et al.* (1975b), comparing different phosphate mixtures, indicated that PSE hams treated with mixtures of tripolyphosphates and metaphosphates had significantly lower Hunter L* values (darker meat), higher pH values and higher final yields compared to hams treated with metaphosphate and non-treated samples (controls). This suggests that certain polyphosphates can improve final yield, as well as other quality characteristics (e.g. colour) of cured/smoked hams from low quality (PSE) meat. In the present investigation the canned hams from all three genotypes were subjected to two treatments: sodium tripolyphosphate (STPP) addition of 0.3% on final product, and no phosphate addition. This was done to determine the effect of genotype on cooking loss in canned hams, and to what extent the added phosphates will be able to overcome the effects of PSE meat, which was prevalent in both the Nn and nn genotype.

8.4.2 Salt

Studies by Mahon (1961) involving the increase in volume of cured meat products indicated that the volume of meat increased as the pH increased (or decreased away from the iso-electric point of pH = 5.5). Addition of low concentrations of salt (0.5%) decreased meat volume. However, meat volume increased with higher salt levels, reaching a maximum at ± 5% salt. This is, however, an unacceptable high level, since most meat products contain between 1.5 and 2% salt. The addition of STPP (0.5%) substantially increased meat volumes, above that of salt alone. This investigation suggested that treatment with only 4% salt gave similar results to treatments with 2% STPP, without added salt. As the permitted phosphate levels differ between countries, it becomes difficult to suggest ideal phosphate/salt levels. However, if processed meat products are to be exported to markets that ban the use of any food phosphates, processors will have to take notice of the complex interactions between factors such as pH and NaCl levels. In the present investigation, salt levels in the finished products (bacon, canned hams, fresh sausage and viennas) ranged from 2 to 2.5%, which is considered an acceptable level of inclusion and is used in the factory where this investigation was done.

8.5 Moisture retention in cured products: use of other additives

Increasing water retention of cured products, such as bacon and ham, is one of the primary functions of brine. High levels of NaCl can result in firmer texture (Hand *et al.*, 1987) but excessive intake of sodium has been related to an increased incidence of hypertension in humans (Dahl, 1972).

The moisture retained with high levels of brine addition is often not satisfactory and processors have used hydrocolloid gelling agents such as carrageenan (an anionic polysaccharide) and starch as binding agents in the manufacturing processes for ham. The functional properties of carrageenan are related to its gel structure formation with meat proteins (Bater *et al.*, 1993), whereas starch is traditionally used to improve quality and extend the higher cost meat fraction of the final product. The effect is based on the ability to gelatinize when heated in a medium containing water, thereby binding relatively large amounts of water (Hodge & Osman, 1976). Starch addition (2%) has been reported to improve surface area, sliceability and texture of hams containing 0.5% carrageenan (Trudso, 1985). However, results from Prabhu and Sebranek (1997) indicated no synergistic effect on moisture retention due to a carrageenan and starch combination. Starch and carrageenan seem to have opposite effects: increased levels of starch increased the perception of juiciness, with increased purge. Addition of carrageenan increased cooking yield and decreased purge, but resulted in a sensory perception of reduced juiciness. In the present investigation rusk was used as an additional moisture retaining additive in the manufacturing of the fresh sausages to determine to what extent such additives would be able to overcome the effect of genotype on WHC and thus product quality.

8.6 Cured meat colour and phosphates

The relationship between PSE and the physical appearance of cured bacon is not so clear. Taylor *et al.* (1973) reported paler bacon when prepared from PSE meat than when prepared from normal meat (measured with a reflectometer) but visible colour differences were only seen in two toned *semitendinosus* muscles. A report by Hall (1950) indicated that polyphosphates with chain lengths of three or more, provide excellent protection against discolouration in products such as frankfurters and whole muscle hams when the pH is maintained between 6.5 and 7.0. However, as soon as the pH is lowered to levels below 6.5, discolouration sets in. The use of polyphosphates in curing salts increases the tolerance of meat products to variations such as cooking temperatures and pH values. Sair and Komarik (1968) reported that if acid reacting phosphates are used to obtain low pH values, highly improved colour stability is obtained with no adverse effects on emulsion formation or stability. In the present investigation CIELAB colour evaluation was done on the back bacon prepared from the different genotypes to determine the effect of genotype on the colour of the final

product. However, comparison of different levels of phosphates on meat colour was not done since this did not fall within the scope of this investigation.

8.7 Meat quality and shrinkage during transporting and processing

Comparison of hams prepared from DFD, normal and PSE meat (that was subjected to transportation prior to processing) indicated that hams prepared from PSE meat had 3-fold greater weight losses (1.51%) than that of hams prepared from normal meat (0.45%) and 7-fold greater losses than hams prepared from DFD meat (0.23%). During subsequent curing and smoking, weight losses hams in prepared for PSE meat were greater (5.71%) than that prepared from both normal (3.95%) and DFD meat (1.64%), thus having an even greater financial impact on profitability than was previously anticipated (Kaufmann *et al.*, 1978).

8.8 Tumbling and massaging

Tumbling and massaging is a method by which mechanical agitation is employed to aid in the rapid diffusion of brine within the muscle (Krause *et al.*, 1978), promoting the formation of a protein exudate which enhances muscle cohesion (Fukazawa *et al.*, 1961a, b). This process makes the muscle more pliable, produces fewer voids, enhances brine retention and improves yield (Torr, 1965). Many researchers attribute the complex binding phenomenon in processed meat products to myofibrillar proteins in general, but Nakayama and Sato (1971) and Samejima *et al.* (1969) reported that myosin and actomyosin contribute to the development of binding. MacFarlane *et al.* (1977) measured binding strength of muscle proteins and their results suggest that myosin exhibits superior binding capabilities to actomyosin in the presence or absence of salt. The heat induced binding (during cooking) between meat pieces is another complex phenomenon, affected by the composition of the exudate and the extent of cellular disruption and breakage which takes place during massaging or tumbling (Siegel *et al.*, 1978).

Gillett *et al.* (1981) showed that increased massaging resulted in an increased distribution of cure ingredients, with a resultant more uniform colour. Krause *et al.* (1978) and Ockerman *et al.* (1978) reported similar results for tumbling. Ockerman *et al.* (1978) reported that short term tumbling (30 min) improved muscle cohesion, but did not significantly affect yield. Apparently a longer time is required to increase yield when compared to cohesion. Motycka and Bechtel (1983) reported no significant differences in yield when comparing continuous tumbling with intermittent tumbling. Results from Krause *et al.* (1978) suggest that shorter tumbling periods may result in brine movement and protein extraction, sufficient to result in acceptable ham quality. The improvement in yield caused by the development of a surface moisture barrier of exudate requires a longer period

of tumbling, interrupted with sufficient rest periods to permit the movement of the salt soluble proteins. Chow *et al.* (1986) studied the main effects; electrical stimulation, rigor condition and tumbling and their interactions on certain quality characteristics of canned pork shoulder. The cooking yield was not influenced by rigor condition, but it increased due to tumbling of prerigor meat when compared to post rigor meat.

8.9 The effect of heating rate and pH on thermal gelation

Gelation of muscle proteins contribute to the desirable texture and stability of fat and water in processed meat products. This is dependent on ionic strength, pH, heating temperature and heating rate, post mortem history of the muscle and muscle type. The latter is a consequence of myosin isoforms, initial pH, protein extractability and functional properties relating to species or fibre type (Samejima *et al.*, 1992). Results from Lan *et al.* (1995) showed that muscle gelation properties of pork, such as force required to rupture gel and force required to move plunger through gel were highest at pH > 6.0 for both slow and fast heating rates. Cooking losses also decreased as the pH increased, suggesting that increased pH (above 6.0) in processed meat would improve protein and water binding.

8.10 Fresh comminuted and emulsion meat products

Genotype in sausage manufacturing is of importance since fresh meat quality has direct bearing on final product quality. Comminuted meat products, such as fresh sausages, have a well established market position and considerable innovation has taken place in recent years to enhance and improve such products. Products that are perceived to be of higher quality are those containing more lean meat and less fat. Internationally legislation exists regulating, among others, lean meat content and it is thus of importance to consider meat quality aspects such as WHC, as it has a considerable influence on the final product. Comminution plays an important role, apart from reducing the particle size of the meat pieces, in extracting salt soluble meat proteins, thus assisting in the binding of the product. It also reduces the obtrusiveness of fat and connective tissue found in such products (Varnam & Sutherland, 1995).

The WHC of the meat used to manufacture sausage has a direct impact on quality because it reduces drip loss from both fresh and frozen sausage, it also reduces cooking loss and retains moisture which adds to the 'juicy' taste of the product. Sair and Cook (1938 as cited by Ellinger, 1972) reported that the minimum amount of drip loss occurred during thawing of ground meats when the pH was ± 6.4, while the maximum amount of drip loss was obtained at a pH of 5.2. Grinding is also associated with an accelerated pH drop (Newbold & Scopes, 1971). Grinding lamb

muscle resulted in a pH decrease from 6.82 to 5.63 within 6 h, compared to a decrease to a pH of 6.46 for the unground controls (Pearson *et al.*, 1973b). Prerigor meat has superior water binding properties and is used for the manufacturing of fresh sausage in the U.S.A. The higher pH values associated with grinding and salting of pre rigor meat may be due to the effect of NaCl and increased chilling rate on the inhibition of post mortem glycolysis, combined with a prolonged aerobic metabolism supported by the oxygen introduced during pre rigor grinding (Judge & Aberle, 1980). The addition of NaCl after comminution presumably solubilizes the protein before strong actomyosin bonds are formed (Varnam & Sutherland, 1995). Attempts at utilising frozen salted pre rigor meat has resulted in a loss of certain functional properties, but this can be overcome by cryostabilisation with non sweetening dextrose polymers.

Some of the more prominent adjuncts added during sausage manufacturing, apart from water, are rusk, casings, NaCl and phosphates. Rusk is added to contribute to sensory aspects such as mouthfeel and texture, while absorbing free moisture. The casing comprise ± 1.0% of the total weight of the sausage and can be broadly classified as artificial or natural. Artificial casings are mostly made from regenerated collagen, with desirable properties such as robustness and "easy to use" application (Varnam & Sutherland, 1995).

8.11 The effect of salt, fat and pH on sensory characteristics of emulsified meat products

Typical emulsified products, such as viennas and frankfurters, usually contain ± 30% fat and less than 2% NaCl. Due to health concerns, the American Heart Association suggested limiting fat consumption to less than 30% of total calories consumed, and sodium (Na) intake to less than 3 g/day (NRC, 1989). However, one frankfurter can contribute more than 14 times that amount of Na (Maurer, 1983). Reduction of Na levels are usually accompanied by adverse changes in physical and sensory properties. Trout and Schmidt (1987) reported that a decrease in salt content from 2.93% to 2.13% and 1.33% respectively, resulted in a forced decrease of water temperature to which the product could be heated, before the water binding ability of the product was negatively affected. However, some water binding abilities that could be lost by reducing salt content may be partially compensated for by altering the pH of the raw meat batter. An investigation by Matulis *et al.* (1995) indicated that acceptable emulsion products, such as viennas and frankfurters, could be manufactured with a minimum of 11.25% fat and 1.3% salt, provided that the pH is equal to 6.0.

8.12 The role of particle size on sensory and physical properties of comminuted products

Results from Small *et al.* (1995) indicated that particle size of ground meat did not affect cooking loss, springiness, cohesiveness or chewiness. Additional mixing tended to decrease sensory particle size and increase springiness and chewiness.

8.13 Connective tissue and phosphates: consequences in emulsion products

Results from Eilert *et al.* (1996), comparing the effects of phosphates (acidic, neutral, alkaline) and modified beef connective tissue in frankfurters, reported lower processing yields and increased colour intensity with acidic phosphates, suggesting that the acidic phosphates might be more effective in direct treatment of high collagen materials in a preblend rather than in direct addition into a frankfurter formulation.

8.14 Conclusion

In conclusion, although certain additives (e.g. phosphates, NaCl) can rectify, to a certain extent, some of the problems (colour, WHC) associated with poor meat quality, it will still result in an inferior product compared to products manufactured from normal (non-PSE, non-DFD) meat. In addition, the economic consequences can be just as severe, with significantly lower net yields, and thus less saleable product, if PSE type meat is used to manufacture value added products (e.g. back bacon).

9 MATERIALS AND METHODS

9.1 Pigs

Landrace x Large White crosses (n = 60) of the three halothane genotypes (NN = 25 , Nn = 19, nn = 16) were sourced from various studs and producers (at approximately 30 kg live weight) in the Western Cape and raised at the University of Stellenbosch piggery till slaughter weight was attained. Since the majority of the pigs slaughtered in the Western Cape are crosses from these two white breeds, this investigation does not endeavour to compare the effect of the halothane gene in the different breeds. No intact males (boars) were used since the local market discriminates heavily against boars due to meat quality deviations associated with boar taint. The genotypes and sexes are specified in Table 9.1.1.

Table 9.1.1 Number and sex of pigs in each genotype.

Sex	Genotype NN	Nn	nn
Castrates	12	7	6
Gilts	13	12	10
Total	25	19	16

9.2 Genotype identification

The pigs either originated from a population of known genotype, or were tested by either the Agricultural Research Centre's Animal Improvement Institute (Irene) or the Department of Biochemistry, University of Stellenbosch using the protocol adapted from the method described by Fujii *et al.* (1991).

9.3 Slaughter procedures

Upon reaching a live weight of ± 86 kg, feed was withheld for 10 h prior to slaughtering, however, water was supplied until loading and transportation. The pigs were weighed again after the 10 hour feed withdrawal period to record empty live weight. No electrical prodders were used to handle the pigs at any stage and loading and transportation was done under conditions of minimal stress (no mixing of unfamiliar pigs, no overcrowding, early morning to avoid high temperatures). Transportation by road to the slaughter facility took approximately 5 min. The pigs were kept in lairage for approximately 1 hour prior to slaughtering.

The pigs were slaughtered following commercial procedures. This consists of electrical stunning (250V AC, ear to ear for 3 - 5 sec) and sticking within 30 sec. Scalding was done by submersion for

approximately 5 min in a water tank at a regulated water temperature of 60°C. Hair was removed by scraping with knives. This was followed by evisceration and inspection of the carcasses by a government health official. After the initial measurements were taken (pH_{45}, warm carcass weight) the carcasses were chilled at 2°C for 24 h where after additional measurements were taken. All the scales used were calibrated prior to commencement of the investigation.

9.3.1 Dressing percentage

Dressing percentage is calculated as the ratio of cold carcass weight to live weight after fasting, and expressed as a percentage. The equation for dressing percentage:

Dressing percentage (%) = (cold carcass weight/live weight) x 100

9.3.2 Chilling loss

Chilling loss is expressed as the percentage weight loss of the carcass during the 24 h chilling period prior to processing of the carcasses. This was calculated as the difference between warm carcass weight (60 min post mortem) and cold carcass weight (24 h post mortem), expressed as a percentage of warm carcass weight. The equation for chilling loss:

Chilling loss (%) = ((warm carcass weight – cold carcass weight)/warm carcass weight) x 100

9.4 Carcass measurements

9.4.1 General remarks

Carcass splitting and cutting techniques differ between countries and different factories within countries. It was therefore decided to follow a technique employed by a major pig abattoir and processing plant in the Western Cape. This particular abattoir split carcasses into primal cuts consisting of shoulders, backs, bellies, legs and front and rear trotters while the exact anatomical positions of cutting depends on customer requirements.

The purpose of determining the composition (lean, bone and fat) of the two primal cuts (leg and shoulder) was to compare the three genotypes and determine what the effect of the presence of the halothane gene on the different components (lean, fat, bone) were. Research (Jones *et al.*, 1988; Fisher *et al.*, 1994; Leach *et al.*, 1996) has indicated that nn carcasses have a higher proportion of lean and a lower proportion of bone and fat compared to NN carcasses, with Nn being intermediate. In this investigation the leg was further subdivided into sub primal cuts (See: # 9.4.4 Deboning technique) to determine which of these cuts (topside, silverside, thickflank and rump) differed the most among genotypes, and which of these cuts make the largest contribution to the difference in yield, either on weight, expressed as a percentage of the primal cut or as a percentage of cold

carcass weight. All the pigs were slaughtered at ± 86 kg live weight, so that direct weight comparisons (between genotypes) of the primal and sub primal cuts can be made. Such results would give a clearer indication of the effect of the halothane gene in specific cuts and show whether the advantages associated with it (higher lean content) translates into higher lean weight.

9.4.2 Cutting and splitting of carcasses

The carcasses were removed from the chiller after 24 h, and each carcass weighed to determine cold carcass weight (see: # 9.5 Linear measurements and # 9.3 Slaughter procedures). The heads were then removed by cutting at a 90 degree angle to the ventral line between the atlas and axis. The carcasses were then transferred to a stationary bandsaw, placed on their backs (feet in the vertical position), with the cranial end of the carcasses facing the cutting edge of the bandsaw blade, and split lengthwise by cutting from the cranial to the caudal end. The lengthwise split was achieved by sawing on an imaginary line parallel to and in the middle of the spinal column. Each half was then resuspended by hanging from the hind foot. Carcass length and certain midline fat measurements were then taken on the exposed medial surface of the left half of each carcass (see: # 9.5 Linear measurements).

9.4.3 Removal of the three primal cuts

The removal of the three primal cuts (leg, shoulder and back) were done on a stationary bandsaw. The left side of each carcass was removed from the suspended position and, after removal of the trotters, prepared for cutting into three primal cuts (leg, back and shoulder). The front foot was removed, using a knife, by cutting in the joint between the lower arm and carpal bones, leaving only the scapula, humerus, radius and ulna on the shoulder primal cut. The rear foot was removed, using a band saw, by cutting through the shinbone (tibia and fibula), halfway between the distal head of the femur and the proximal head of the tibia. Although this position was not measured, the inaccuracy that may have occurred here was counteracted by the deboning technique applied (see: # 9.4.7 Deboning technique)

9.4.3.1 Leg

The leg was removed by cutting in a line perpendicular to the spinal column, between the 5^{th} and 6^{th} lumbar vertebra.

9.4.3.2 Shoulder

The shoulder was removed by cutting in a line perpendicular to the spinal column, cranially between the first and second cervical vertebrae (the atlas and axis), and caudally through the 5-6^{th}

thoracic vertebrae. By employing this technique, the entire blade bone (scapula) with adhering connective tissue remains on the shoulder cut and no part of this can be detected in the primal back cut.

9.4.3.3 Back

The backs from both the left and right sides of the carcasses were used. Both backs were removed from the carcass, cranially at a line perpendicular to the spinal column, between the 6th and 7th thoracic vertebrae and caudally at a line perpendicular to the spinal column, between the last lumbar and first sacral vertebrae. The bellies were removed from the backs by cutting in a line parallel to the spinal column, approximately 18 cm from the spinal column. The right side backs were used for manufacturing bacon (see: # 9.7 Manufacturing techniques). The left side backs were used to remove certain portions for determining drip loss, fat thickness and muscle depth measurements, colour measurements, cooking loss and sensory evaluation (see: # 9.5 Linear measurements, # 9.6.3 Colour measurements and # 9.8 Sensory evaluation).

9.4.4 Deboning technique

The deboning of the cuts described below was done in such a manner that the minimum meat remained on the bones, although no claims can be made that no visible meat remained after deboning. Six deboners were employed and trained by the same master butcher, who supervised every deboning session.

9.4.4.1 Leg

The left hind leg of each carcass was dissected into four sub primal cuts, namely the rump, silverside, topside and thickflank. Deboning proceeded by placing the leg on its lateral side (Figure 9.4.4.1.1), opening and removing the skin and subcutaneous fat on the medial side of the leg as close as possible to the muscles, thus revealing the topside *(M. biceps femoris*, *M. gracilis*, and adductors of the thigh and part of the *M. quadriceps*). The skin and fat was removed up to the contact point on the table (Figure 9.4.4.1.2). The vertebrae were removed by two cuts close to the bone. The visible part of the pelvic bone that remained, attached to the femur, was cleaned of any visible muscle (lean trimmings) and then removed (Figure 9.4.4.1.3). The topside was separated from the thickflank (Figures 9.4.4.1.4 and 9.4.4.1.5) by cutting on the second visible line of the top layer of muscles on the ventral surface between the *M. biceps femoris* and the *M. quadriceps femoris* (part of the thickflank), leaving the head of the *M. psoas* on the topside. The topside was then cut loose from the full medial surface of the femur and separated from the top surface of the silverside, above the 'eye' of the silverside (*M. semimembranosus*). The *M. gastrocnemius* does not

form part of the silver or topside in South Africa and is left on the lower leg. The femur was removed from the distal end, leaving the thickflank, rump, silverside and lower leg clearly visible (Figure 9.4.4.1.6). The lower leg was then separated (including the *M. gastrocnemius*) and removed from the silverside. The thickflank (including the patella) was separated from the silverside and rump, followed by the separation of the rump and silverside. The silverside was then removed from the subcutaneous fat and skin, and trimmed of all excess fat. The lower leg was then deboned, removing the tibiotarsus (Figure 9.4.4.1.7). All the weights of the primal cuts, fat/skin, lean trimmings and bones were recorded individually. The top and silverside of each individual animal was then wrapped in polyethelyne bags, placed in boxes and frozen (-40°C). These cuts were subsequently defrosted for 24 h after all the pigs used in the investigation were slaughtered, and used to manufacture processed products (see: # 10.7 Processed products).

9.4.4.2 Shoulder

The left shoulder of each pig was dissected into fat/skin, bone and lean meat. Deboning was done by placing the shoulder on its lateral side, loosening the subcutaneous fat and skin visible on the medial side, and proceeding to the lateral side by turning the shoulder over. The defatted and skinned shoulder was then once more placed on its lateral side, and the ulna, radius, scapula, the first and second cervical vertebrae as well as the 1st to 5th thoracic vertebrae (with ribs attached) were then removed. All the weights of the fat/skin, bone and lean meat were recorded individually.

9.4.4.3 Back

The right side back, used for bacon manufacturing, was deboned by removing the remaining lumbar and thoracic vertebrae, and remaining part of the ribs. The subcutaneous fat/skin was also removed so that a fat layer of approximately 0.5 cm remained. All the weights were recorded individually.

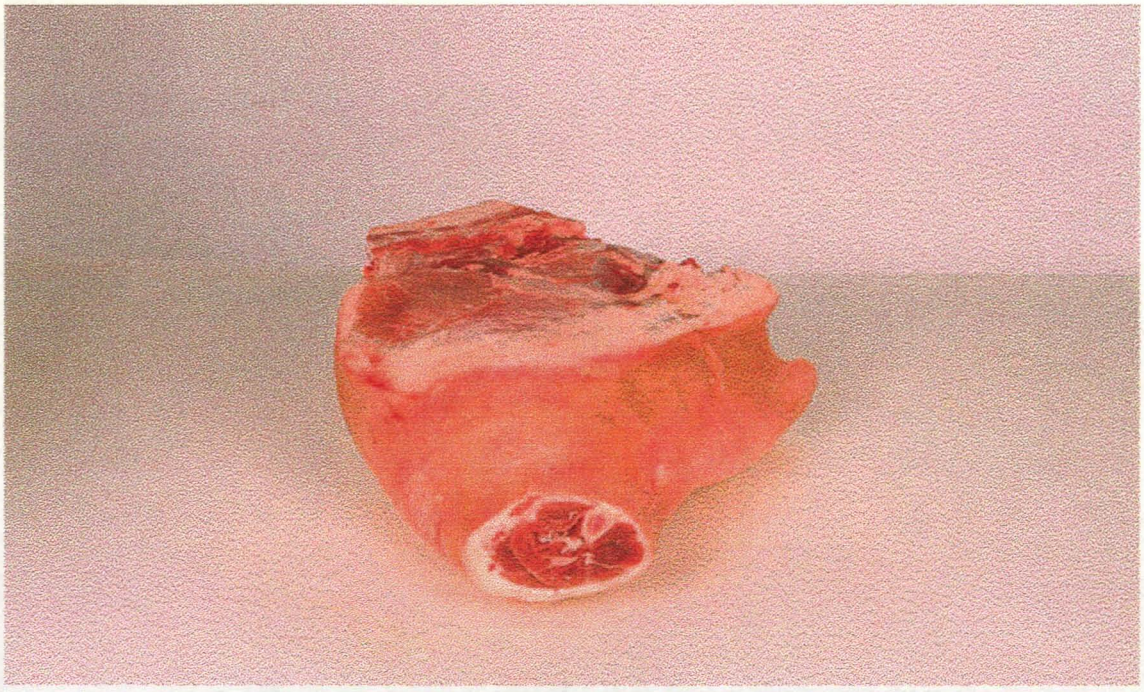

Figure 9.4.4.1.1 Intact leg viewed from medial angle

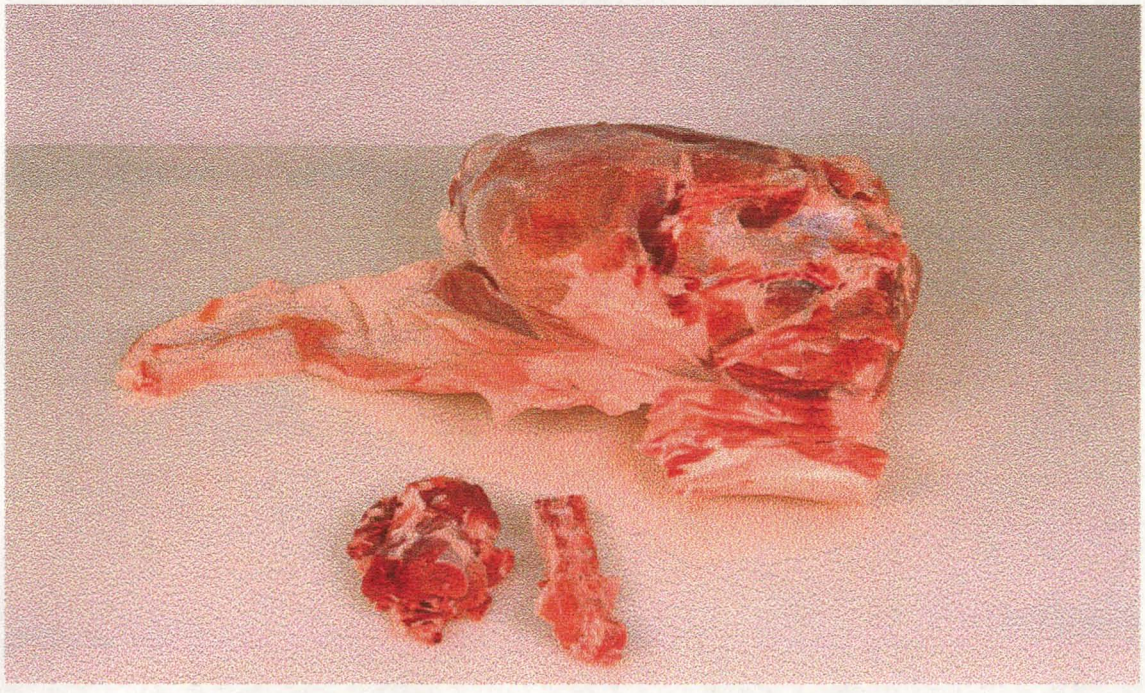

Figure 9.4.4.1.2 Fat and skin removed up to contact point, vertebrae and lower leg removed

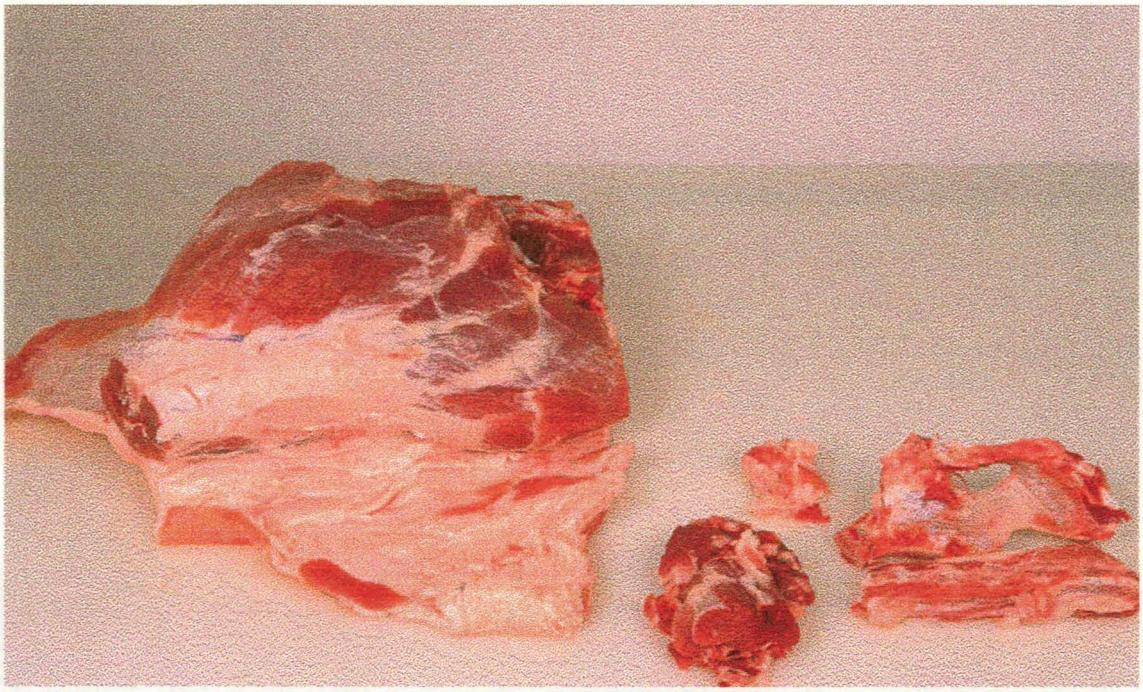

Figure 9.4.4.1.3 Pelvic bone removed

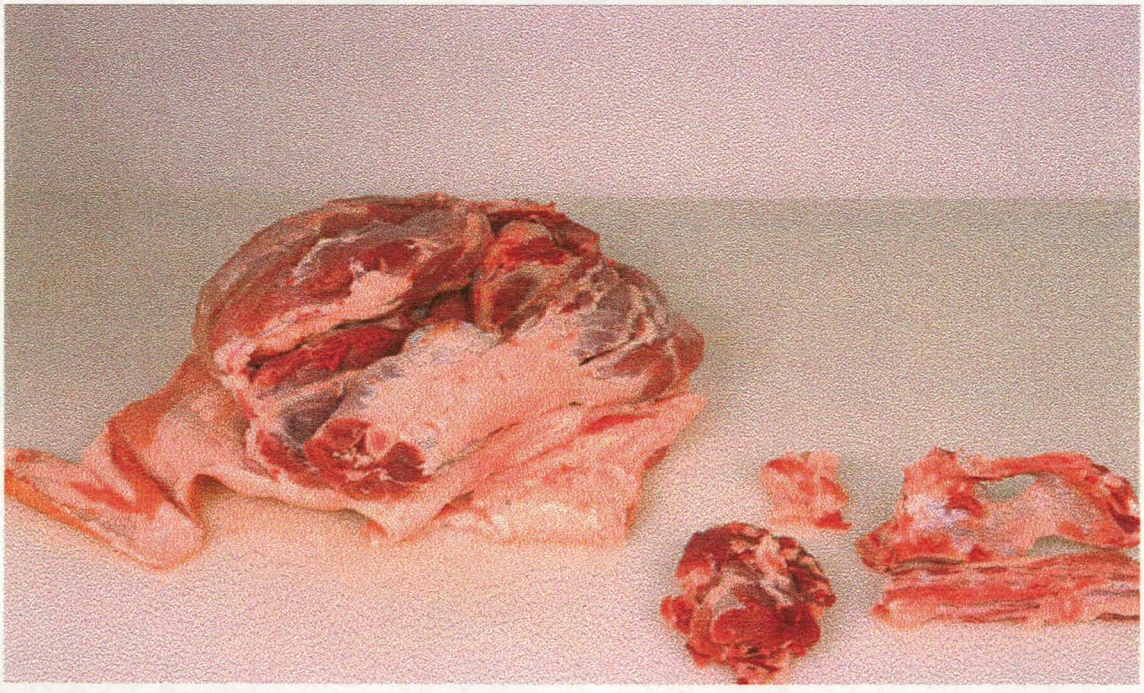

Figure 9.4.4.1.4 Topside separated from thickflank

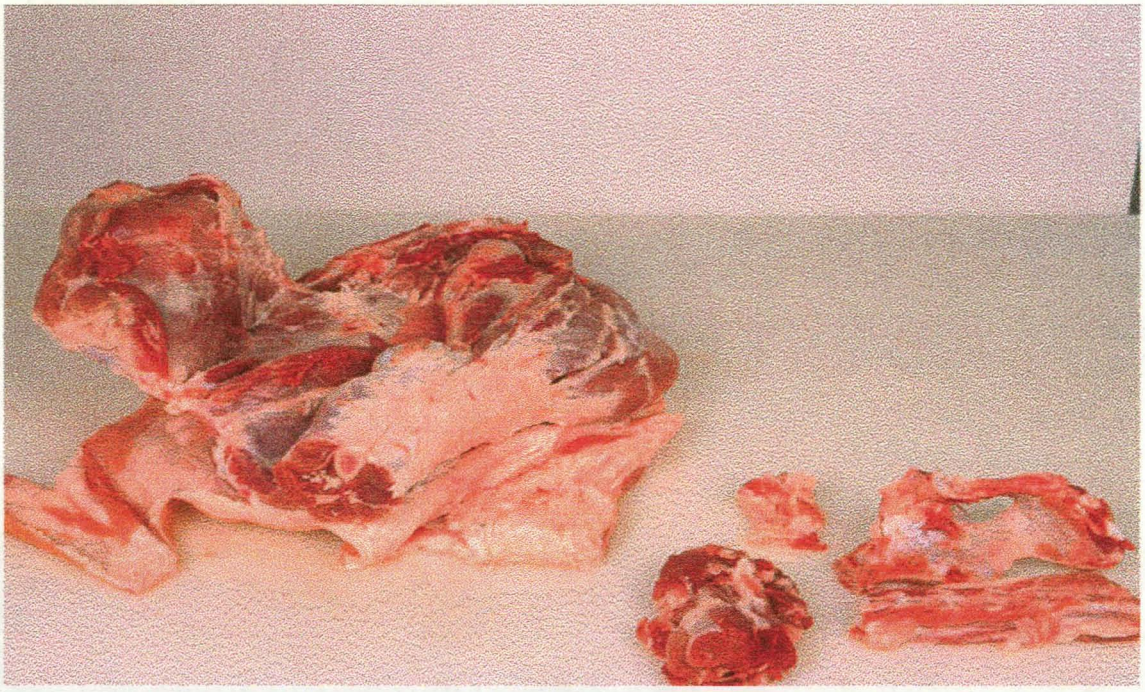

Figure 9.4.4.1.5 Topside separated from thickflank, exposing the silverside

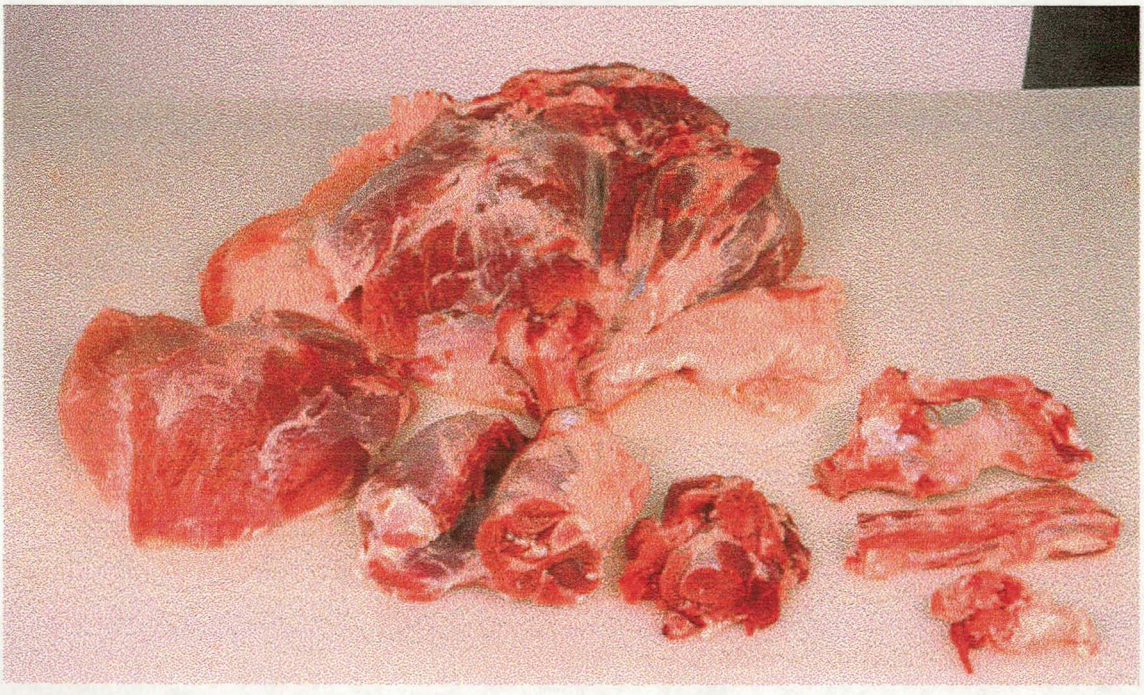

Figure 9.4.4.1.6 Topside, lower leg and femur removed

Figure 9.4.4.1.7 Anti clockwise from top left: silverside, topside, lean trimming, lower leg lean, thickflank and rump. Femur, pelvic bone, tibiotarsus and patella visible in foreground

9.5 Linear measurements

The presence of the halothane gene (Nn, nn) is associated with increased MLT measurements and reduced backfat measurements, compared to NN genotypes (Jones *et al.*, 1988; Fisher *et al.*, 1994). Depending on the regression formulae (and the independent variables) used to determine percentage predicted lean yield, this normally leads to higher predicted carcass lean yields for the Nn and nn genotypes.

9.5.1 Carcass length

Carcass length was measured as the distance, in mm, from the cranial tip of the first thoracic vertebra (viewed medial) to the nearest cranial tip of the pelvic bone. All carcass length measurements were done with a metal ruler.

9.5.2 Length:live weight ratio

The length:live weight ratio was calculated by dividing the carcass length (See: # 9.5.1 Carcass length) with the live weight (after fasting). This ratio was calculated to compare genotypes and sexes and to determine if differences in carcass length between genotypes or sexes was due to weight, genotypic or sex differences. The equation of length:live weight ratio:

Length:live weight ratio = live weight (kg)/carcass length (cm)

9.5.3 Midline fat measurements

After splitting the carcass lengthwise, the subcutaneous fat thickness of the left medial side of each carcass was measured (in mm) at different anatomical locations. The measurement positions (as defined by the spinal vertebrae or visible muscles) were: opposite the first thoracic vertebra, between the 2nd and 3rd last thoracic vertebrae and in the middle of the visible portion of the *M. gluteus medius*. All midline fat measurements were done with a metal vernier calliper calibrated in mm.

9.5.4 Percentage predicted lean, fat thickness and muscle depth measurements

The left side back of each carcass was used to determine certain fat thickness and muscle depth measurements. A slice, 25 mm thick, was removed anterior of the 3rd last rib (so that measurements could be taken on a position between the 2nd and 3rd last rib) and placed on a flat surface. To determine percentage predicted lean yield (LMP) as measured with the Hennessey Grading Probe (HGP), a subcutaneous fat thickness and the eye muscle depth (MLT) was measured 45 mm from the midline, perpendicular to the centre of the thoracic vertebra. These measurements were recorded in mm. The maximum depth (mm) of the MLT was also measured over the largest distance

perpendicular to the centre of the thoracic vertebra, approximately 60 mm from the midline, and the width over the widest distance perpendicular to the axle used to determine depth. The latter measurements were also recorded in mm, while all measurements were done with a metal vernier calliper. Percentage predicted lean (LMP) was calculated from the fat thickness and muscle measurements taken on the position between the 2nd and 3rd last rib, 45 mm from the midline using the following formula (Government Notice No. R. 1748, 26 June 1992):

$$\text{HGP: } LMP = 72.5114-(0.4618 \times FT) + (0.0547 \times MD)$$

9.5.5 Muscle area

The area of the MLT for each carcass was measured on the 25 mm thick slice used for determining fat thickness and muscle depth. The area was determined by placing a glass plate over the slice, tracing the outline of the visible portion of the MLT and thereafter measuring the area with a calibrated planimeter. Values are given as cm^2.

9.6 Meat quality

Various reports (Jones *et al.*, 1988; Murray *et al.*, 1989; De Smet *et al.*, 1992; Fisher *et al*, 1994) indicate that the presence of the halothane gene (either in Nn or nn form) contributes significantly to a decline in meat quality characteristics such as pH, colour and drip loss, especially when present in the homozygous form (nn). There are currently no standards for pork meat quality in South Africa, and a number of breeders and producers have included the gene in producing heterozygotes (Nn), which is believed to be beneficial for producing higher carcass lean yields, as determined in the present South African classification system. This system does not take into account meat quality or the incidence of PSE meat. One of the purposes of this investigation was to quantify the differences in meat quality between the three genotypes, and to determine whether these differences are of such a nature that recommendations can be made as to the inclusion or exclusion of the halothane gene in local production conditions.

9.6.1 pH

The pH values (pH_{45} and pH_{24}) of the MLT were measured using a hand held Crison pH/mV-506 meter equipped with a glass electrode. Before use, the pH meter was calibrated in pH 4 and pH 7 buffers. The pH meter was recalibrated after every fourth reading and the electrode rinsed with distilled water between measurements. The measuring point on the carcass was defined as a point between the 2nd and 3rd last thoracic vertebra, 45 mm from the midline. Two measurements per carcass were recorded: pH_{45} approximately 45 min after slaughter, and pH_{24} measured on the cold

carcass 24 h after slaughter. Insertion depth of the probe into the MLT at each reading was 3 cm. Both measurements (pH_{45} and pH_{24}) were made at a similar position, but taking care not to measure in the exact same position, thus avoiding any possible variation due to the insertion of the probe into the meat and subsequent exposure of the meat to the surrounding atmosphere.

9.6.2 Drip loss

After splitting the carcasses lengthwise, a slice 25 mm thick, was removed from the anterior end of the left side of each back at the position of the 6th thoracic vertebra. The samples were weighed individually and placed, under atmospheric pressure, in a net enclosed in a plastic bag in such a manner that the exudate did not come into contact with the sample, but was collected in the bag. The samples were stored for 48 h at 2°C. Drip loss is reported as the weight loss expressed as a percentage of the original weight of the sample. The samples were touch dried with a paper towel prior to weighing after the 48 h period elapsed.

Drip loss (%) = ((fresh sample weight – sample weight after 48 h)/fresh sample weight) x 100

9.6.3 Colour measurements

Colour was evaluated using a Colorgard System 2000 colorimeter (Pacific Scientific, Silver Spring, MD, USA) to determine CIELAB values (L*, a* and b* values), with L* indicating brightness, a* the red-green range and b* the blue-yellow range. Fresh meat colour was determined on the same sample (see: # 9.5.4 Percentage predicted lean, fat thickness and muscle depth measurements) used for measuring muscle area and fat and muscle thickness. The colour evaluation on the back bacon (see: # 9.7.1 Preparation of back bacon) was determined with the similar technique on a sample taken by cutting a slice from the centre portion of the back, approximately 25 mm thick.

9.6.4 Cooking loss and shear value

Cooking loss (%) was determined by placing weighed MLT samples of approximately 100 g, sealed in plastic bags, in water at 75°C for 50 min. This allowed for sufficient heat penetration without causing excessive denaturation of collagen present in the meat. The bagged samples were allowed to cool down in running water to ± 25°C. Weight of the cooked samples were determined after decanting the liquid phase, and the cooking loss calculated as total fluid lost, expressed as a percentage of the fresh (uncooked) sample. Three 1.27 cm diameter samples (from the centre of each MLT sample) were randomly removed for determining Warner-Bratzler shear force values (Voisey, 1976). The samples were cut parallel to the muscle fibre direction in order to measure the influence of the myofibrillar proteins. Maximum shear force values (kg/1.27 cm) were recorded for each sample (repeated three times) and a mean was calculated for each individual animal.

9.6.5 Chemical analyses

Proximate analysis (protein, moisture, fat and ash) of fresh MLT samples (with the visual subcutaneous fat removed) was carried out according to AOAC standard techniques (AOAC, 1984). All samples were oven-dried (110°C for 24 h). Protein content was determined by the Kjeldahl method and fat content by ether extraction.

9.7 Manufacturing techniques

The manufacturing techniques used in this investigation were similar to those that are currently practised in a major meat processing plant in the Western Cape, and may differ from techniques used in other countries to manufacture similar products. Results from Fisher and Mellett (1997) indicated that the presence (or absence) of the halothane gene resulted in measurable differences in processing yields and losses for certain manufactured pork products under commercial or factory conditions. This investigation was done to determine the influence of the gene on a range of products such as fresh manufactured pork products (fresh sausage), whole muscle cured and smoked pork (back bacon), canned pork (canned hams), as well as emulsified pork (viennas). An sensory evaluation was also included to determine if a trained sensory panel would be able to identify genotypes on aspects such as colour and juiciness. Such results can possibly be used to identify alternative uses for PSE meat (which is associated with the presence of the halothane gene) in manufactured pork products.

9.7.1 Preparation of back bacon

Back bacon is a raw, cured (added NO_2) product that is prepared from the deboned and partially defatted "backs" of pig carcasses ("back" referring to the total length of the MLT, from the 5^{th} or 6^{th} thoracic vertebrae up to the last lumbar vertebra). These are usually prepared fresh (unfrozen) within 24 h after slaughter. The width of the cut varies, but is usually on average 18 cm wide, measured from the midline of the back (medial side) towards the belly (ventral side). It is a high priced product in South Africa and should contain 95% total meat by chemical analyses (Foodstuffs, Cosmetics and Disinfectants Act, Act 54 of 1972). After curing and smoking at temperatures of 68°C, this processed cut is sliced to 2 to 3 mm slices (called rashers) and is usually packed in neatly shingled vacuum packs of 250 or 500 g. The appearance of the product is raw and it is usually fried in oil before consumption, served as a breakfast product, usually 1 to 2 rashers per serving, with fried eggs and toasted bread.

In this investigation, a procedure practised by a well known factory in the Western Cape, and producer of a popular brand of bacon was followed. This comprises the following: preparation of a

curing brine containing salt (sodium chloride), sodium nitrite, sodium tripolyphosphate (STPP), dextrose, ascorbic acid and water. The composition of the brine is presented in Table 9.7.3. This brine is usually injected into the MLT (Belam multi-needle brine injector, calibrated to inject 20% by weight at an operating pressure of 2 bar) to what is known in the industry as 120%, i.e. 20 kg brine is injected into 100 kg of meat. These levels are hardly ever achieved with automated equipment, and to compensate for any differences that may result from inefficient injection, the backs are placed in an identical "cover brine" at a ratio of 100 kg raw (uninjected) meat to 20 kg brine for a 12 to 24 h period. Thereafter the individual backs are hung on metal hooks, placed in smoke racks and cold smoked (with Meranti wood chips) in a Maurer smoker at 50°C for 2 h. After subsequent chilling to an internal temperature of at least –4°C, the nett yield should be approximately what is known as 105% (100 kg meat yields 105 kg bacon). Under these circumstances the legal requirements of 95% meat (minimum) is met.

In the present investigation the exact procedures were followed as practised in the industry, except the backs were kept under frozen storage (– 40°C, individually wrapped in polyethelene bags and placed in cardboard boxes for 2 months) prior to processing, since it was more important to process all products together rather than as individually as the pigs reached slaughter weight. Several weight recordings were made. Five weight losses were recorded for each back during processing: frozen weight, thawed weight, weight immediately after brine injection, weight after tank curing and weight after completion of the smoking and chilling process (net weight). The following four values were then calculated for each back, thaw loss (%), pumped gain (%), brine loss (%) and total gain (%). The thaw loss was calculated as the percentage loss in moisture after thawing, pumped gain as the percentage gain in weight after brine injection, brine loss as the percentage moisture or brine loss after tank curing and total gain as the percentage net gain after completion of the process. The backs were cut in half perpendicular to the median (spinal column) and a slice (1 cm thick, weighing approximately 120 g) was removed for CIELAB colour analyses.

9.7.2 Preparation of comminuted products

After thawing for 24 h, the top and silverside of each individual carcass were grouped according to genotype. These pooled cuts (consisting of top and silverside) from the different genotypes were then minced, using a 32 mm diameter mincing plate. After thorough mincing the meat of the three genotypes were subdivided into batches for the manufacturing of canned hams, fresh sausages and viennas.

9.7.3 Canned hams

To determine the effect of the addition of phosphates on differences between genotypes on the processing characteristics of canned hams, two 10 kg batches of each genotype (two treatments per genotype; 0% and 0.3% phosphate addition rate, calculated on the finished product) were prepared for processing. The brine mixture (Table 9.7.3.1) for each treatment was dissolved in water, added to the meat and the latter tumbled continuously for 25 min. This is a relatively short tumbling cycle, but was chosen to emphasise possible differences between genotypes.

After tumbling each ham mixture was canned (300 g per can, 20 cans per treatment), sealed and sterilised at 115°C for 50 min in an autoclave. To determine cooking losses the cans were stored for one month before removing the contents. The meat of each can was removed, carefully dried with absorbent tissue and weighed. Cooking loss is expressed as the difference in weight after sterilization and storage, as a percentage of the weight prior to sterilization. The cooking loss equation is:

Cooking loss (%) = ((fresh weight – cooked weight)/fresh weight) x 100

Table 9.7.3.1 Brine ingredients for bacon and ham products

	Ingredients (% in brine)			Ingredients (kg/100 kg meat)		
	Bacon	[1]Ham (P)	[2]Ham	Bacon	[1]Ham (P)	[2]Ham
Phosphate	2.0	1.5	0	0.4	0.3	0
NaCl	10.0	12.5	12.5	2.0	2.5	2.5
$NaNO_2$	0.1	0.1	0.1	0.02	0.02	0.02
Dextrose	5.0	4.5	4.5	1.0	0.9	0.9
Ascorbate	0.25	0.25	0.25	0.05	0.05	0.05
Water	82.65	81.15	82.65	16.53	16.23	16.53
Meat	-	-	-	100	100	100
Total	100	100	100	120	120	120

[1]Ham (P): ham with phosphate
[2]Ham : ham without phosphate

9.7.4 Fresh pork sausage

For the preparation of the fresh sausages the sub sample of meat of each genotype (see: 9.7.2 Preparation of comminuted products) was minced a second time using a 5 mm diameter mincing plate. Two 7.5 kg batches of each of the genotypes were prepared, one with 5 % rusk added, the

other without rusk. Table 9.7.4.1 reflects the ingredients used to manufacture the sausages. For each batch, the ingredients were added to cold water and thoroughly mixed prior to adding to the minced meat. The meat was then placed in a bowl cutter and cut for approximately 15 sec, whilst the ingredients were added. The temperature of chopped meat ingredients in the cutter was constantly monitored and stayed below 5°C. The mixtures were then transferred to a Handmann sausage filler (model FA 30), calibrated to fill links into 65 g portions. Collagen sausage casings (Devro) were used in an effort to minimize the risk of case rupturing during the manufacturing process.

After completion of the filling process, the sausages were divided into bundles of 10 sausages per bundle, 10 bundles for each of the two treatments (5% rusk, no rusk) within each genotype. The weight of each bundle was determined after preparation where after it was hung to dry for 24 h at 2°C. After drying the bundles were weighed to determine chilling loss, with the amount of chilling loss expressed as:

chilling loss (%) = ((fresh weight - chilled weight)/fresh weight) x 100.

After weighing, the sausages were packed in styrofoam trays, 5 sausages per tray, and wrapped with polyethylene. The wrapped sausages were then placed back into cold storage (2°C) until commencement of the sensory trails.

Table 9.7.3 Ingredients of sausage type products

	Ingredients (kg/100 kg meat)		
	[1]Sausage (R)	[2]Sausage	Vienna
Rusk	5.0	0	0
NaCl	1.5	1.5	1.8
$NaNO_2$	0	0	0.02
Dextrose	0.9	0.9	0.9
Ascorbate	0.05	0.05	0.05
Water	12.55	17.55	17.23
Meat	100	100	100
Total	120	120	120

[1]Sausage (R): sausage with rusk
[2]Sausage : sausage without rusk

9.7.5 Viennas

For the preparation of the viennas the sub sample of meat of each genotype (see: # 9.7.2 Preparation of comminuted products) was minced using a 5 mm diameter mincing plate and placed in a bowl

cutter where the ingredients (Table 9.7.4.1) were added and the mixture cut until it resembled a fine paste. The emulsion temperature was constantly monitored and maintained below 5°C. The emulsion was placed in a Handmann sausage filler (model FA 30), calibrated to fill links into 65 g portions. Collagen sausage casings (Devro) were used in an effort to minimize the risk of case rupturing during the manufacturing process.

After completion of the filling process the viennas were divided into bundles of 10 viennas per bundle, 10 bundles for each of the three genotypes. The bundles were weighed individually (fresh weight) and hung on a smoke rack. The viennas were dry smoked (using Meranti wood chips) for 60 min at 60°C. After smoking the viennas were cooked to an internal temperature of 68°C, and allowed to dry before chilling (2°C) for 24 h. The bundles were weighed (cooked weight) and the weight difference (cooking loss) was expressed as:
cooking loss (%) = ((fresh weight - cooked weight)/fresh weight) x 100.

9.8 Sensory evaluation

The sausages, that were packed and wrapped (each sample consisted of five sausages), were kept in cold storage (2°C) for seven days until commencement of the taste evaluation. The tasting was done over five days with two taste evaluations per day to avoid fatigue of panellists. All the treatments were evaluated during each tasting. To avoid bias, each genotype (within each treatment) was assigned a random assorted three digit number (Snedecor & Cochran, 1980). Since design of the trail precludes comparison of the two treatments (5% rusk, no rusk) the panelists were informed which samples included or excluded rusk.

On commencement of each tasting the sausages were removed from cold storage, the wrapping removed and the weight recorded to determine cooking loss. In this trail it was anticipated that some sausages may appear lighter (pink) than others, and that others may appear darker (also pink). However, the effect of oxygenation of the myoglobin pigment (to oxymyoglobin), the visibility of the pigment and other physical properties, such as particle size, concealed the distinct pinkness of the product. Some sausages appeared slightly grey in colour due to the original state of the visible myoglobin. Even so, the panellists were requested to rank the raw sausages according to colour intensity (dark pink, pink, light pink), comparing genotypes within each treatment (rusk and no rusk). These results are presented in a bar chart (See: Figures 10.7.3.2.1 and 10.7.3.2.2), with each bar indicating the number of times panellists ranked a sample in one of the three classes (the design of the experiment precludes any statistical analysis of these results).

The sausages were cooked on a grill in an oven for 12 min and at 210°C until a core temperature of 73 - 78°C was reached. The samples were evenly distributed in the oven to overcome possible variation in oven temperatures at different locations. Five minutes after removal from the oven, cooked weights were recorded to determine cooking loss expressed as:
cooking loss (%) = ((fresh weight - cooked weight)/fresh weight) x 100.

The samples were presented in randomly numbered trays. The cooked sausages were evaluated for juiciness comparing genotypes within each treatment (5% rusk and no rusk). Five descriptive classes were used (very dry, dry, neither dry nor juicy, juicy, very juicy) and each panellist was required to indicate which word was the most descriptive of each cooked sample. Subsequently, the frequency for each class was calculated and statistically analysed.

9.9 Statistical analyses

9.9.1 Null hypothesis and test of significance

Analysis of variance were performed on all the variables measured using the General Linear Models (GLM) procedure of SAS (Statistical Analysis System Institute, Inc., 1988). A full model was used to determine the presence of two way interaction. The model is depicted below:

$Y_{ij} = \mu + G_i + S_j + GS_{ij} + e_{ij}$

Y_{ij} = Dependent variable of the *i*th genotype of the *j*th sex

μ = Overall mean

G_i = Genotype effect

S_j = Sex effect

GS_{ij} = Interaction between genotype and sex

e_{ij} = Random error of measurement term

The differences between genotypes and sexes were, where appropriate, tested separately by means of the null hypothesis (H_o), with $H_o: \mu = \mu_o$ and the alternate hypothesis (H_a) being $H_a: \mu \neq \mu_o$. This was done by means of contrast analyses and the estimated least square means (± standard error) are reported in the tables contained in the text. Differences between dependent variables for genotypes and sexes are reported significant if the probability of rejection of $H_o < 5\%$.

9.9.2 Correlation

The correlation coefficient (r) measures the degree of closeness of the linear relationship between two variables (Snedecor & Cochran, 1980). If one variable (e.g. X_1) can be expressed as an exact linear function of another variable (e.g. X_2), then the correlation is +1 or -1, depending on the nature of the relationship, being direct (+1) or inverse (-1). Another property of r is that it is without unit or dimension since the units of its numerator and denominator are both the products of the units in which X_1 and X_2 are measured. SAS determines the true product correlation P (Pearson) and is defined as:

$P_{xy} = \text{cov}(x,y)/\sqrt{[\text{var}(x)\ \text{var}(y)]}$

The true correlation is estimated by the sample correlation r (Pearson product moment) ,and is calculated as:

$r_{xy} = \Sigma(x_i-x)\ (y_i-y)/\sqrt{[\_x_i-x)^2\ \Sigma(y_i-y)^2]}$

where x and y are the sample means of X and Y.

The significance level of the correlation is indicated as PROB > |R| (SAS: users guide: Basics, 1988). It is important to note that even if convincing evidence of an association does exist, it does not prove that x is the cause of the variation in y and that evidence of the causality must come from other sources.

9.9.3 Multiple linear regression

Regression values (R^2) were calculated using the SAS RSQUARE procedure. R^2 is the square of the multiple correlation coefficient and, when expressed as a percentage, gives an indication of the percentage variation in the dependent variable as explained by the independent variables. If a model with multiple parameters is fitted, then

R^2 = 1 - [Residual Sum of Squares/Total Sum of Squares]

This is used as a summary measure of the quality of the fitted model, a higher R^2 indicating a higher regression and thus a more accurate predictive model. The Cp-statistic (Mallows, 1973) was used for selecting suitable models for predicting the dependent variables. This method uses the full model with all the variables as a base of reference and it makes adjustments for the number of variables included. Possible drawbacks are that this statistic assumes the selected model to be the full model, taking into account all possible variables that may affect the dependent variable. This, of course, is not always the case. Secondly, this statistic lacks a significance test to compare different subsets. Mallows defines

$Cp = RSS_p/\delta^2 + 2p\text{-}n$

RSS_p = Residual Sum of Squares where only p variables are included in the model.
δ^2 = Residual Mean Square for the full model.
n = number of observations.
p = number of variables in subset

If there is no bias in the chosen model, then Cp should be on average equal to p. Thus, choosing a suitable model would be to plot Cp against p and choose the simplest model with Cp near to but preferably less than p.

9.9.4 Analyses of ordinal data

The data was analysed in the style of the cheese tasting experiment in section 5.6 of McCullagh and Nelder (1989), using PROC LOGISTIC in the SAS system. Two contrasts were evaluated, the first comparing the juiciness of sausage prepared from meat of the NN genotype to that from the nn genotype. The second contrast compared the juiciness of the Nn genotype to the mean of the juiciness of the NN and nn genotypes, asking, in effect, whether this juiciness is halfway between the juiciness observed for the homozygote. These contrasts were done on both treatments, with 5% rusk and without rusk added.

10 RESULTS AND DISCUSSION

10.1 Carcass quality

The presence of the halothane gene, in Nn and nn genotypes, is associated with carcasses that have a lower fat content and a higher lean yield, compared to the genotype that does not have the halothane gene (Jones *et al.*, 1988; Murray *et al.*, 1989; Fisher *et al.*, 1994).

10.2 Carcass weights

The results for live, warm and cold carcass weight and dressing percentage are given in Table 10.2.1. Live weights were the lowest for the NN genotypes (84.9 kg) and the highest for the nn genotypes (87.5 kg) , with Nn intermediate (86.1 kg). There were no significant differences for any of the genotypic or sex comparisons. The results from this investigation indicate that dressing percentage did not differ significantly between the homozygotes (NN and nn), although the Nn genotypes had significantly higher values ($P < 0.05$). The relatively small differences in dressing percentage between genotypes suggests that the live weight differences can be disregarded. Results from Jones *et al.* (1988) show that the nn genotypes had significantly higher dressing percentages compare to the NN and Nn genotypes in a live weight range of ± 100 kg, which was ascribed to smaller proportions of of body organs, alimentary tracts, fat depots and gutfill. The results from this investigation do not offer conclusive proof that the presence of the halothane gene will increase dressing percentage in the 80 – 90 kg live weight range in such a manner that it will significantly increase economic returns for pork producers. It must be noted that this parameter can be influenced by factors such as breed, diet, management prior to slaughter and the definition of dressing percentage which differs depending on which country the investigation was conducted in.

10.3 Midline carcass measurements

The three midline (1st thoracic vertebra, between the 2nd and 3rd last rib and on the *M. gluteus medius*) fat thickness measurements (Table 10.2.1) suggest a progressive decline in fat thickness in the presence of the halothane gene. Although the weight differences between genotypes and sexes were non - significant ($P > 0.05$), it was initially thought that the apparent higher live and carcass weights (Table 10.2.1) of the Nn and nn genotypes, compared to the NN genotypes, would result in decreased differences between genotypes with regard to carcass measurements, such as fat thickness and carcass length, since both increase with an increase in live and carcass weight.

The nn genotypes had the lowest values for fat thickness for all three midline fat measurements, differing significantly from the NN genotypes ($P < 0.05$) on the T_{2-3} position and on the *M. gluteus medius*. As expected, differences between sexes were significant ($P < 0.05$) for all three midline fat

measurements, with the gilts having the least amount of midline fat. In contrast to the present investigation, other reports (Jones *et al.*, 1988; Leach *et al.*, 1996) have not given such clear differences between the different genotypes.

Carcass lengths were lowest for the NN genotypes (763 mm), differing ($P < 0.05$) from the nn genotypes (778 mm), which had the highest value, with the Nn genotypes (773 mm) having an intermediate value. Various studies (Eikelenboom *et al.*, 1980a; Andresen *et al.*, 1981; Jones *et al.*, 1988; Simpson & Webb, 1989) show that the nn genotype have shorter carcasses than NN genotypes, with Nn genotypes showing an intermediate position. Pommier *et al.* (1992), however, showed no significant differences in carcass length between NN and Nn genotypes. However, it must be kept in mind that the live weights in the above mentioned studies ranged from approximately 85 kg to over 110 kg, which could influence gene expression with regard to certain carcass traits. The length:live weight ratios were calculated to determine whether the higher values for carcass lengths of the Nn and nn genotypes were due to increased live and carcass weights (Table 10.2.1). It appears that the increased live and carcass weight of the Nn and nn genotypes, although not significant, could have contributed to the longer carcass lengths, since length/live weight ratios did not differ ($P > 0.05$) between either genotypes or sexes. Differences in carcass lengths did not differ significantly between the sexes and genotype x sex interaction was absent.

Table 10.1.1 The effect of genotype and sex on carcass quality characteristics (least square mean ± std. error)

	Genotype			Sex		Gen x Sex interaction
	NN	Nn	nn	Barrow	Gilt	P > t
Live weight (kg)	84.9 ± 0.694	86.1 ± 0.824	87.5 ± 0.895	86.6 ± 0.724	85.8 ± 0.590	0.559
Warm carcass weight (kg)	65.5 ± 0.728	67.7 ± 0.865	67.6 ± 0.939	67.4 ± 0.760	66.5 ± 0.619	0.785
Cold carcass weight (kg)	64.1 ± 0.735	66.0 ± 0.873	66.3 ± 0.948	65.9 ± 0.767	65.0 ± 0.624	0.669
Carcass length (mm)	763^{a} ± 4.29	773^{ab} ± 5.10	778^{b} ± 5.53	771 ± 4.48	772 ± 3.64	0.693
Length/live weight ratio	8.98 ± 0.058	9.00 ± 0.069	8.90 ± 0.075	8.91 ± 0.061	9.01 ± 0.049	0.699
Dressing percentage	77.1^{a} ± 0.433	78.7^{b} ± 0.515	77.3^{a} ± 0.559	77.8 ± 0.443	77.5 ± 0.375	0.973
Midline fat measurements (mm)						
1st thoracic vertebra (T_1)	32.4 ± 0.925	30.3 ± 1.10	29.5 ± 1.19	32.4^{a} ± 0.966	29.1^{b} ± 0.786	0.568
2^{nd}-3^{rd} last rib (T_{2-3})	20.4^{a} ± 0.748	19.4^{ab} ± 0.888	17.5^{b} ± 0.956	20.7^{a} ± 0.781	17.5^{b} ± 0.636	0.464
M. gluteus medius (GM)	19.6^{a} ± 0.885	18.4^{ab} ± 1.05	16.0^{b} ± 1.14	19.4^{a} ± 0.924	16.6^{b} ± 0.752	0.834

[a-c] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.05$), according to contrast analyses.

[d-f] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.001$), according to contrast analyses.

Table 10.1.1 (continued) The effect of genotype and sex on carcass quality characteristics (least square mean ± std. error)

	Genotype			Sex		Gen x Sex interaction
	NN	Nn	nn	Barrow	Gilt	P > t
2nd-3rd last rib measurements (mm) , 45 mm off midline (T_{2-3} - 45 mm).						
Fat depth	$17.4^{a} \pm 0.802$	$16.4^{ab} \pm 0.952$	$13.7^{b} \pm 1.03$	16.5 ± 0.837	15.2 ± 0.681	0.919
MLT depth	$54.3^{a} \pm 0.934$	$57.9^{b} \pm 1.11$	$64.5^{c} \pm 1.20$	59.1 ± 0.975	58.7 ± 0.793	0.658
MLT measurements (mm) at 2nd-3rd last rib						
MLT width	$85.8^{a} \pm 1.08$	$90.0^{b} \pm 1.28$	$94.9^{c} \pm 1.39$	89.7 ± 1.12	90.8 ± 0.915	0.915
MLT depth	$55.8^{d} \pm 0.931$	$58.6^{d} \pm 1.11$	$65.5^{e} \pm 1.20$	60.0 ± 0.972	59.9 ± 0.791	0.254
MLT area (cm^2)	$35.2^{d} \pm 0.950$	$40.3^{e} \pm 1.13$	$46.4^{f} \pm 1.22$	40.2 ± 0.991	41.0 ± 0.807	0.799
Percentage predicted lean (LMP)	$67.5^{a} \pm 0.397$	$68.1^{a} \pm 0.471$	$69.7^{b} \pm 0.512$	68.1 ± 0.414	68.1 ± 0.338	0.882

a-c Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.05$), according to contrast analyses.

d-f Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.001$), according to contrast analyses.

10.4 Lateral carcass measurements and predicted lean yield

In this investigation the presence of the halothane gene (Table 10.1.1) was accompanied by lower fat thickness and higher eye muscle depth values, measured between the 2nd and 3rd last ribs, 45 mm from the midline (T_{2-3}-45 mm). The NN genotypes had higher ($P < 0.05$) fat thickness (17.3 mm), lower ($P < 0.05$) eye muscle depth (54.4 mm) and lower percentage predicted lean yield (67.5%) compared to the nn genotypes which have the leanest carcasses (69.8%) calculated from the fat thickness (13.6 mm) and eye muscle depth (64.5 mm). The Nn genotypes had intermediate values for fat thickness (16.3 mm), eye muscle depth (57.6 mm) and predicted lean yield (68.2%). There was no genotype x sex interaction.

The MLT measurements (width, depth and area) were taken at the same position, between the 2nd and 3rd last ribs, as those for calculating percentage lean yield in the carcass. However, the eye muscle depth was taken as the largest distance over the MLT perpendicular to the centre of the thoracic vertebra and the width at the largest distance perpendicular to the axis used to determine depth. MLT area was determined with a calibrated planimeter, as described earlier (see: # 9.5.5 Muscle area). The measurements taken on the MLT had a similar pattern to those taken during carcass classification (T_{2-3} - 45 mm) with the nn genotypes having the largest MLT area (46.4 cm^2), confirmed by the width and depth measurements. These measurements differed significantly for the nn genotypes ($P < 0.001$) from the Nn and NN genotypes (40.3 cm^2 and 35.2 cm^2, respectively), with the NN genotypes exhibiting the smallest MLT area ($P < 0.001$).

Comparison of the sexes did not show any significant differences for fat thickness or loin eye depth on the T_{2-3}-45 mm position, and subsequently the calculated lean yield differences between barrows (67.8%) and gilts (68.6%) were also not significant. However, the results do suggest that the gilts had a tendency towards leaner carcasses with less fat. The results of this investigation indicate that the gilts had larger eye muscle dimensions (width and depth) and areas compared to the barrows (41.0 cm^2 and 40.2 cm^2, respectively), although none of the differences were significant ($P > 0.05$). Genotype x sex interaction was absent.

Similar to these results, a number of studies (Webb *et al.*, 1994; Leach *et al.*, 1996) have indicated that there were small or no differences between NN and Nn genotypes with regard to length and midline fat measurements. However, the midline measurements in this investigation suggests that the presence of the halothane gene is accompanied by a reduction in midline fat thickness, results similar to that of De Smet *et al.* (1992), although other investigations have shown no significant reduction in fat thickness (Jones *et al.*, 1988; Simpson & Webb, 1989). The gilts had consistently

less fat (Table 10.2.1) than the barrows, with no difference in either carcass length or length/live weight ratio. This is consistent with previous findings that gilts produce leaner carcasses (measured at both the midline and lateral points) than barrows (Jones *et al.*, 1988; Sather *et al.*, 1989).

Current carcass evaluation systems (including the South African system) are based on lateral fat and muscle thickness measurements. This is due to (apart from practical considerations) the fact that the lateral measurements (over the MLT) are more accurate predictors of percentage lean yield than midline measurements (Fortin *et al.*, 1984). The MLT measurements for the three genotypes are consistent with findings in the literature (Jones *et al.*, 1988), identifying the nn genotypes with the largest MLT areas. However, Jones *et al.* (1988) pointed out that the lower backfat measurements of the nn genotypes, which tend to have shorter and thicker muscled carcasses, may not identify their superior lean yield compared to NN genotypes, even if the fat thickness measurements were similar. Since one of the most important reasons for inclusion of the halothane gene is increased percentage lean yield (based on scientific results), such shortcomings in any classification system would neutralise any possible advantages associated with the halothane gene (Jones *et al.*, 1988; Sather *et al.*, 1991a; Pommier *et al.*, 1992; Fisher *et al.*, 1994). As the results from this investigation indicate, these advantages in lean yield are more than offset by the serious decline in meat quality e.g. meat colour, drip and moisture loss (see: # 10.6 Meat quality).

10.5 Primal cut distribution and cutting yields.

10.5.1 Shoulder yield

Examination of the results in Table 10.5.1.1 indicate significant interaction between sex and genotype for bone yield from the shoulder, whether this yield was expressed as weight (Table 10.5.1.2 and Table 10.5.1.3), a percentage of the shoulder (Table 10.5.1.4 and Table 10.5.1.5), or as a percentage of the cold carcass weight (Table 10.5.1.6 and Table 10.5.1.6). The apparent tendency for increased bone yield with inclusion of the halothane gene, as in Table 10.5.1.1, is therefore examined further by investigating this interaction in Table 10.5.1.3, Table 10.5.1.5 and Table 10.5.1.7. Since bone is a non-saleable by-product, or of extremely low value, this apparently similar weight yield for gilts and castrates, and apparently increasing tendency for higher bone yield with inclusion of the halothane gene is of interest, since other investigations (Jones *et al.*, 1988) have indicated lower proportions of bone (expressed as a proportion of of the total weight of the primal cuts) with inclusion of the halothane gene.

Genotypic comparison (Table 10.5.1.1) show that the nn genotypes had the heaviest bone content of the three genotypes (NN = 1269 g, Nn = 1327 g and nn = 1348 g), with a similar pattern emerging if expressed as a percentage of total shoulder weight (NN = 16.6%, Nn = 17.1% and nn = 17.3%) and as a percentage of cold carcass weight (NN = 3.96%, Nn = 4.02% and nn = 4.06%). None of the genotypic comparisons show statistically significant differences, which could be due to the nn genotypes having slightly heavier total shoulder weights than the other two genotypes, thus also showing increasing bone content. In contrast, Jones *et al.* (1988) reported that the nn genotype had the lowest proportional bone yield, with NN the highest and Nn intermediate. However, in this investigation, none of the differences between sexes or genotypes showed any statistical significance ($P > 0.05$).

Further examination of the within genotype comparison revealed that the biggest difference in bone yield (Table 10.5.1.3) between sexes was for the NN genotype (castrates = 1188 g, gilts = 1349 g), with nn the lowest (castrates = 1383 g, gilts = 1312 g) and Nn intermediate (castrates = 1381 g, gilts = 1274 g), with a similar pattern being shown when expressed as a percentage of shoulder weight (Table 10.5.1.5) and as a percentage of cold carcass weight (Table 10.5.1.7).

Table 10.3.1.1 The effect of genotype and sex on fat/skin, bone and lean yield from the shoulder (least square mean ± std. error).

	Genotype			Sex		Gen x Sex interaction
	NN	Nn	nn	Barrow	Gilt	P > t
Weight, g						
Total	7647 ± 109	7779 ± 129	7758 ± 140	7836 ± 114	7620 ± 92.6	0.223
Fat/skin	1289^{a} ± 45.6	1227^{ab} ± 54.2	1092^{b} ± 58.8	1268^{a} ± 47.6	1137^{b} ± 38.7	0.548
Bone	1269 ± 29.4	1327 ± 34.9	1348 ± 37.9	1318 ± 30.7	1312 ± 25.0	0.008
Lean	5079^{a} ± 75.8	5208^{ab} ± 90.1	5298^{b} ± 97.8	5201 ± 79.3	5162 ± 64.4	0.567
Percentage of total shoulder weight, %						
Fat/skin	16.8^{d} ± 0.513	15.8^{de} ± 0.610	14.1^{e} ± 0.662	16.2 ± 0.536	14.9 ± 0.436	0.859
Bone	16.6 ± 0.264	17.1 ± 0.314	17.3 ± 0.341	16.8 ± 0.276	17.2 ± 0.225	0.005
Lean	66.5^{a} ± 0.538	66.9^{ab} ± 0.639	68.7^{b} ± 0.694	66.9 ± 0.562	67.9 ± 0.457	0.085
Percentage of cold carcass weight, %						
Total shoulder	23.88 ± 0.217	23.57 ± 0.257	23.39 ± 0.279	23.79 ± 0.226	23.43 ± 0.184	0.273
Fat/skin	4.02^{d} ± 0.130	3.72^{de} ± 0.154	3.29^{e} ± 0.167	3.85 ± 0.135	3.50 ± 0.110	0.683
Bone	3.96 ± 0.0765	4.02 ± 0.0909	4.06 ± 0.0987	3.99 ± 0.0812	4.04 ± 0.0650	0.006
Lean	15.87 ± 0.173	15.77 ± 0.205	16.07 ± 0.223	15.91 ± 0.180	15.89 ± 0.147	0.425

$^{a-c}$ Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.05$), according to contrast analyses (see text).
$^{d-f}$ Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.001$), according to contrast analyses (see text).

Although the gilts yielded lighter shoulders (P > 0.05) compared to the castrates (Table 10.5.1.1), the actual bone yields (g) were very close to that of the castrates. These results suggest that the gilts yielded a higher percentage of bone in this particular cut, both when expressed as a percentage of shoulder weight (Table 10.5.1.1) and as a percentage of cold carcass weight. These results are in support of Jones *et al.* (1988) who indicated that for all the primal cuts, castrates had a significantly lower proportion of bone, when expressed as a proportion of the primal cut.

Table 10.5.1.2 ANOVA for shoulder bone yield (g)

Source	Df	SS	MS	F	Pr > F
Sex	1	8063.4	8063.4	0.37	0.543
Genotype	2	42815.6	21407.8	0.99	0.378
Interaction	2	226995.7	113497.9	5.26	0.008
Error	54	1165331.8	21580.2		
Total	59	1443206.6			

Table 10.5.1.3 Means for shoulder bone yield (g)

Sex	NN	Nn	nn	WM[1]
Castrates	1188	1381	1383	1318
Gilts	1349	1274	1312	1312
WM[2]	1269	1327	1348	

[1] Weighed means for sex
[2] Weighed means for genotypes

Table 10.5.1.4 ANOVA for shoulder bone yield as a percentage of shoulder weight

Source	Df	SS	MS	F	Pr > F
Sex	1	6.12	6.12	3.50	0.067
Genotype	2	3.56	1.93	1.10	0.339
Interaction	2	20.27	10.12	5.81	0.005
Error	54	94.27			
Total	59	124.52			

Table 10.5.1.5 Means for shoulder bone yield as a percentage of shoulder weight

Sex	NN	Nn	nn	WM[1]
Castrates	15.7	17.5	17.2	16.8
Gilts	17.5	16.7	17.4	17.2
WM[2]	16.6	17.1	17.3	

[1] Weighed means for sex
[2] Weighed means for genotypes

Within sex comparisons indicate that for castrates, inclusion of the halothane gene resulted in a higher bone yield (Table 10.5.1.3), with nn the highest (1383 g), Nn intermediate (1381 g) and NN the lowest (1188 g), showing a similar pattern when expressed as a percentage of shoulder weight (Table 10.5.1.5: NN = 15.7%, Nn = 17.5% and nn = 17.2%) and as a percentage of cold carcass weight (Table 10.5.1.7: NN = 3.72%, Nn = 4.15% and nn = 4.12%). In contrast, the results for gilts (Table 10.5.1.3) indicate that the NN genotypes had the highest bone yield (1349 g), with nn intermediate (1312 g) and Nn the lowest (1274 g). A similar pattern emerged for bone yield as a percentage of shoulder weight (Table 10.5.1.5: NN = 17.5%, Nn = 16.7% and nn = 17.4%) and as a percentage of cold carcass weight (Table 10.5.1.7: NN = 4.20%, Nn = 3.90% and nn = 3.99%).

The results of this investigation suggest that the presence of the halothane gene, in both Nn and nn genotypes, could overshadow the effect of gender (gilt or castrate), which is much more prominently expressed in the NN genotype.

Table 10.5.1.6 ANOVA for shoulder bone yield as a percentage of cold carcass weight

Source	Df	SS	MS	F	Pr > F
Sex	1	1.43	1.43	0.98	0.328
Genotype	2	3.59	0.18	0.12	0.884
Interaction	2	16.51	8.25	5.65	0.006
Error	54	78.84			
Total	59	97.14			

Table 10.5.1.7 Means for shoulder bone yield as a percentage of cold carcass weight

Sex	NN	Nn	nn	WM[1]
Castrates	3.72	4.15	4.12	3.99
Gilts	4.20	3.90	3.99	4.04
WM[2]	3.96	4.02	4.06	

[1] Weighed means for sex
[2] Weighed means for genotypes

Total shoulder weight, expressed as a percentage of cold carcass weight did not show any significant difference among genotypes or sexes. However, there was a declining tendency in this variable with inclusion of the halothane gene. For lean yield and fat/skin yield, no genotype x sex interaction was noted (Table 10.5.1.1). Lean yield increased significantly ($P < 0.05$) from NN (5079 g) to the nn genotype (5298 g), with Nn being intermediate (5208 g), which is supported by a number of reports (Webb *et al.*, 1985; Jones *et al.* 1988). A similar pattern emerged when lean yield is expressed as a percentage of shoulder weight, but fails to reach statistical significance when expressed as a percentage of cold carcass weight. Regardless of expression (weight, percentage of total shoulder weight or as a percentage of cold carcass weight), fat/skin content was the lowest for the nn genotypes ($P < 0.001$ for both percentage of shoulder weight and percentage of cold carcass weight; $P < 0.05$ for fat/skin on weight), differing significantly from the NN genotype. The Nn genotype was intermediate for all three expressions, and did not differ significantly from both NN and nn genotypes.

The gilts show a tendency for higher lean and lower fat and skin content in the shoulder, but this only reached significance ($P < 0.05$) for fat and skin yield (and not when expressed as percentages), with the castrates having significantly more fat and skin (1268 g) compared to the gilts (1137 g). As noted, the presence of the halothane gene is accompanied by a significant increase in lean and a reduction in fat/skin in the shoulder. Gender differences were small and in most cases not significant. The results from this investigation show that the advantageous effect of the halothane gene on carcass composition (higher lean and lower fat content) is reflected in the shoulder joint, as well as the leg joint (see: # 10.5.2 Leg yield). The advantage to both the producer and processor is obvious since the current South African classification system will identify the higher lean yields (Table 10.2.1), based on fat thickness and muscle depth on the classification point. However, as other authors have noted (Jones *et al.*, 1988), there is concern that a single backfat measurement may not identify the superior lean yield of the nn genotype to complete satisfaction.

10.5.2 Leg yield

The results in Table 10.5.2.1 show that for the leg, there was no interaction between sex and genotype. The direct comparison of genotypes and sexes is therefore statistically justified. The composition of the leg (Table 10.5.2.1) indicates that the nn genotypes had a significantly higher ($P < 0.05$) lean yield which was maintained when calculated as a percentage of leg weight and as a percentage of cold carcass weight when compared to the other two genotypes, which did not differ from each other. The heterozygous (Nn) genotype had the highest bone content. Not all the differences between genotypes, expressed on weight and as a percentage of cut or carcass, were significant. However, the nn genotypes had significantly higher ($P < 0.05$) total leg yields (expressed as a percentage of cold carcass weight) compared to the NN and Nn genotypes. This, together with the non significant tendency for a declining proportion of total shoulder (as a percentage of cold carcass weight) for nn genotypes would suggest that the presence of the halothane gene could lead to a difference in certain carcass composition traits (shoulder, leg) with an increase in higher priced primal cuts. The scientific literature indicates conflicting results, with no difference in bone yield between genotypes (Leach *et al.*, 1996) to significant genotypic differences (Aalhus *et al.*, 1991; Sather *et al.*, 1991a) being noted. Bone yield, calculated as a percentage of total leg weight, suggest that the nn genotypes in the present investigation had the lowest bone component. Fat/skin content had a similar trend to that found in the shoulder, with the nn genotypes having significantly less fat ($P < 0.05$) than the NN genotypes, with the heterozygotes being intermediate ($P > 0.05$). The gilts had significantly more lean ($P < 0.05$) when calculated as a percentage of leg weight and less fat/skin ($P < 0.05$) when expressed as a percentage of cold carcass weight. The results from other investigations (Jones *et al.*, 1988; Fisher *et al.*, 1994) show similar trends, with little difference in total leg yield between genotypes.

The results for sub primal lean cuts of the leg are presented in Table 10.5.2.2. No genotype x sex interaction was present. The nn genotypes have, similar to the trend for total lean yield in the leg, the highest yields for all six cuts, with NN the lowest and Nn intermediate in most cases. Not all the differences between genotypes were significant ($P > 0.05$) but when expressed as a percentage of cold carcass weight, most of the cuts (except lower leg lean) were significantly higher ($P < 0.05$) than the other two genotypes. Sex differences were less prominent with only the silverside, both as a percentage of leg weight and cold carcass weight, being significantly higher ($P < 0.05$) for the gilts. The composition of total bone in the leg is shown in Table 10.5.2.3. The heterozygotes (Nn) had the heaviest femur and tibiotarsus, both as a weight and as a percentage of total leg weight. The nn genotypes tend to have, when expressed as a percentage of total leg and cold carcass weight, the

lowest proportion of bone of all three genotypes. This also contributes to the higher lean yield of the leg by causing a more advantageous bone to lean ratio. This may have been partially due to breeders selecting breeding stock based on visual conformation and dorsal fat thickness measurements in an effort to identify individuals with a higher lean meat content. Better conformation carcasses tend to have higher lean to bone ratios and thus more lean at equal fatness than poorer conformation carcasses (Kempster *et al.*, 1982). Thus, as the halothane gene is associated with a better conformation and higher lean yield (Webb & Simpson, 1986), selection of breeding stock on the above mentioned criteria could have led to, initially unknowingly, increased selection for the halothane gene. From the present investigation it is clear that the nn genotypes yield more lean in the dissected shoulder and leg primal cuts than the NN genotypes. This is an advantage to meat processors, since lean meat is of higher value than fatty tissue or bone, provided that the processing characteristics are superior or equal to the other genotypes.

Table 10.5.2.1 The effect of genotype and sex on fat/skin, bone and lean yield of the leg (least square mean ± std. error).

	Genotype			Sex		Gen x Sex interaction
	NN	Nn	nn	Barrow	Gilt	P > t
Leg weight (g)						
Total	7422[a] ± 99.5	7653[a] ± 118	8106[b] ± 128	7743 ± 104	7711 ± 84.6	0.629
Fat/skin	1676 ± 46.7	1598 ± 55.4	1542 ± 60.2	1681[a] ± 48.7	1529[b] ± 39.6	0.581
Bone	636[a] ± 12.3	686[b] ± 14.1	669[ab] ± 15.4	646 ± 12.8	670 ± 10.8	0.986
Lean	5037[a] ± 98.8	5312[a] ± 114	5837[b] ± 124	5226 ± 116	5418 ± 98.4	0.893
Percentage of total leg weight, %						
Fat/skin	22.7[a] ± 0.069	21.0[ab] ± 0.822	19.1[b] ± 0.892	21.8 ± 0.722	20.0 ± 0.588	0.861
Bone	8.57[ab] ± 0.143	8.94[a] ± 0.170	8.21[b] ± 0.184	8.44 ± 0.149	8.72 ± 0.121	0.779
Lean	67.7[a] ± 0.674	69.0[a] ± 0.800	71.9[b] ± 0.856	68.5[a] ± 0.703	70.5[b] ± 0.572	0.807
Percentage of cold carcass weight, %						
Total leg	23.20[a] ± 0.247	23.19[a] ± 0.293	24.48[b] ± 0.319	23.52 ± 0.258	23.73 ± 0.210	0.535
Fat/skin	5.23[a] ± 0.141	4.83[ab] ± 0.168	4.66[b] ± 0.182	5.10[a] ± 0.148	4.72[b] ± 0.120	0.592
Bone	1.99 ± 0.0372	2.07 ± 0.0442	2.01 ± 0.0480	1.98 ± 0.0388	2.07 ± 0.0316	0.943
Lean	15.74[a] ± 0.275	16.15[a] ± 0.327	17.60[b] ± 0.355	16.10 ± 0.287	16.80 ± 0.234	0.699

[a-c] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.05$), according to contrast analyses (see text).
[d-f] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.001$), according to contrast analyses (see text).

Table 10.5.2.2 The effect of genotype and sex on wholesale lean cuts of the leg (least square mean ± std. error).

	Genotype			Sex		Gen x Sex interaction
	NN	Nn	nn	Barrow	Gilt	P > t
Leg, lean weight (g)						
Topside	1386[a] ± 35.9	1473[ab] ± 42.6	1574[b] ± 46.3	1459 ± 37.4	1496 ± 30.5	0.997
Silverside	1626[a] ± 34.5	1755[b] ± 41.0	1908[c] ± 44.6	1725 ± 36.1	1801 ± 29.4	0.719
Thickflank	920[a] ± 22.2	921[a] ± 26.3	1021[b] ± 28.6	940 ± 23.1	967 ± 18.8	0.967
Rump	324[a] ± 19.8	329[a] ± 23.4	432[b] ± 25.6	353 ± 20.7	370 ± 16.8	0.204
Lean trim	143[a] ± 14.4	160[a] ± 17.1	225[b] ± 18.6	181 ± 15.1	171 ± 12.3	0.344
Lower leg lean	635 ± 20.1	647 ± 23.9	669 ± 25.9	654 ± 21.0	647 ± 17.1	0.763
Lean cuts as a percentage of leg weight, %						
Topside	18.6 ± 0.352	19.2 ± 0.418	19.4 ± 0.454	18.8 ± 0.367	19.3 ± 0.299	0.782
Silverside	21.9[a] ± 0.277	22.9[b] ± 0.330	23.5[b] ± 0.358	22.3[a] ± 0.290	23.3[b] ± 0.236	0.683
Thickflank	12.4 ± 0.184	12.0 ± 0.219	12.6 ± 0.237	12.1 ± 0.192	12.5 ± 0.156	0.947
Rump	4.36[a] ± 0.243	4.24[a] ± 0.288	5.34[b] ± 0.313	4.52 ± 0.253	4.77 ± 0.206	0.174
Lean trim	1.92[a] ± 0.190	2.11[a] ± 0.226	2.78[b] ± 0.245	2.31 ± 0.198	2.23 ± 0.161	0.272
Lower leg lean	8.55 ± 0.227	8.44 ± 0.269	8.25 ± 0.292	8.45 ± 0.237	8.38 ± 0.193	0.458

[a-c] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.05$), according to contrast analyses (see text).

[d-f] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.001$), according to contrast analyses (see text).

Table 10.5.2.2 (continued) The effect of genotype and sex on wholesale lean cuts of the leg (least square mean ± std. error).

	Genotype			Sex		Gen x Sex interaction
	NN	Nn	nn	Barrow	Gilt	P > t
Lean cuts as a percentage of cold carcass weight, %						
Topside	4.33[a] ± 0.101	4.46[ab] ± 0.120	4.75[b] ± 0.131	4.44 ± 0.106	4.59 ± 0.086	0.934
Silverside	5.08[a] ± 0.095	5.32[a] ± 0.113	5.76[b] ± 0.123	5.24[a] ± 0.081	5.53[b] ±0.099	0.915
Thickflank	2.88[a] ± 0.59	2.79[a] ± 0.071	3.08[b] ± 0.077	2.86 ± 0.062	2.97 ± 0.051	0.913
Rump	1.02[a] ± 0.062	0.99[a] ± 0.074	1.31[b] ± 0.080	1.08 ± 0.065	1.14 ± 0.053	0.191
Lean trim	0.45[a] ± 0.044	0.49[a] ± 0.053	0.68[b] ± 0.057	0.549± 0.046	0.527± 0.038	0.271
Lower leg lean	1.98 ± 0.059	1.96 ± 0.070	2.02 ± 0.076	1.99 ± 0.061	1.99 ± 0.050	0.700

[a-c] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.05$), according to contrast analyses (see text).

[d-f] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.001$), according to contrast analyses (see text).

Table 10.5.2.3 The effect of genotype and sex on bone yield of the leg (least square mean ± Std. error).

	Genotype			Sex		Gen x Sex interaction P > t
	NN	Nn	nn	Barrow	Gilt	
Leg, bone weight (g)						
Femur	287[a] ±5.11	311[b] ± 6.07	296[ab] ± 6.52	295 ± 5.33	301 ± 4.34	0.597
Pubis	170 ± 5.90	178 ± 7.01	189 ± 7.61	177 ± 6.16	181 ± 5.01	0.716
Tibiotarsus	178[a] ± 5.43	195[b] ± 6.45	182[ab] ± 7.01	180 ± 5.67	190 ± 4.61	0.183
Bone weight as a percentage of leg weight, %						
Femur	3.88[a] ± 0.068	4.07[b] ± 0.081	3.65[c] ± 0.088	3.82 ± 0.071	3.91 ± 0.058	0.920
Pubis	2.29 ± 0.069	2.33 ± 0.082	2.32 ± 0.089	2.28 ± 0.072	2.35 ± 0.059	0.680
Tibiotarsus	2.40[ab] ± 0.066	2.55[a] ± 0.079	2.24[b] ± 0.085	2.33 ± 0.069	2.46 ± 0.056	0.062
Bone weight as a percentage of cold carcass weight, %						
Femur	0.898 ± 0.016	0.943 ± 0.019	0.894 ± 0.020	0.890 ± 0.016	0.928 ± 0.014	0.554
Pubis	0.532 ± 0.018	0.540 ± 0.022	0.569 ± 0.023	0.538± 0.019	0.557 ± 0.015	0.559
Tibiotarsus	0.556 ± 0.016	0.590 ± 0.018	0.548 ± 0.020	0.546± 0.016	0.584 ± 0.013	0.201

[a-c] Values in the same row with the same caption (genotype or sex) with different superscripts differ (P < 0.05), according to contrast analyses (see text).
[d-f] Values in the same row with the same caption (genotype or sex) with different superscripts differ (P < 0.001), according to contrast analyses (see text).

10.6 MEAT QUALITY

10.6.1 pH (pH_{45} and pH_{24})

Comparison (Table 10.6.1.1) of the initial pH values (pH_{45}) show a significant difference ($P < 0.001$) between the three genotypes with the NN genotypes having the highest mean pH_{45} ($pH_{45} = 6.22$). Using initial pH values above or equal to 5.9 to distinguish between normal or PSE meat, the pH_{45} of the NN genotypes are considered normal, with only 8% (Figure 10.6.1.1) of the NN carcasses considered as PSE ($pH_{45} < 5.9$). The choice of an initial pH of 5.9 to discriminate between normal and PSE meat was based on values given in literature (Wismer-Pedersen, 1959a; Cheah *et al.*, 1995, Wenzlawowicz *et al.*, 1996) and thus merely serves as a guideline to compare the meat quality results from this investigation with published reports. The halothane positive (nn) genotypes had the lowest mean pH_{45} value ($pH_{45} = 5.35$), with none of the individual pH_{45} values in this genotype being higher than 5.9, thus identifying all the nn carcasses in this investigation as PSE. The implication of these results are that, if a meat quality classification system based on pH

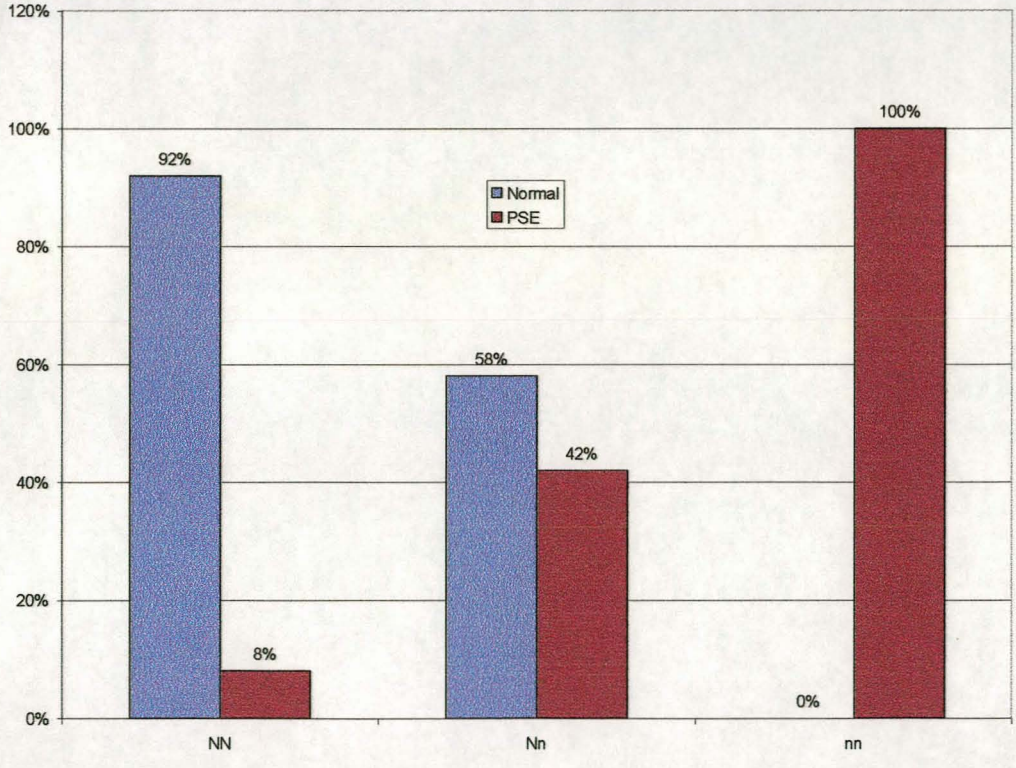

Figure 10.6.1.1 Percentage normal ($pH_{45} \geq 5.9$) and PSE ($pH_{45} < 5.9$) meat within each genotype

was employed in a commercial abattoir, all of these nn genotypes would have been discriminated against on the basis of supposed poor meat quality being associated with a low initial pH. This could mean that any financial gains of a higher carcass lean yield (Table 10.2.1) may be nullified by a reduced premium for meat quality.

The Nn genotypes had an mean initial pH value (pH_{45} = 5.94) intermediate to the NN and nn genotypes, with 42% of the Nn carcasses considered as PSE (pH_{45} < 5.9). Genotype x sex interaction was absent, with no significant differences between sexes (Table 10.6.1.1). The expression of the halothane gene in this characteristic is therefore due to genotype only. The mean ultimate pH values (pH_{24}) showed that the NN genotypes had the highest pH_{24} (pH_{24} = 5.62), differing only ($P < 0.05$) from the nn genotypes, which had the lowest value (pH_{24} = 5.44). The Nn genotypes had an intermediate value (pH_{24} = 5.60), significantly higher ($P < 0.05$) than the nn genotypes. Sex differences were not significant and no genotype x sex interaction was observed (Table 10.6.1.1).

The results from this investigation confirms that the presence of the halothane gene is associated with lower initial pH values, especially so with the nn genotype (Murray *et al.*, 1989; Oliver *et al.*, 1993; Fisher *et al.*, 1994; Fisher, 1995). The large variation in meat quality observed in the Nn genotypes (42% PSE / 58% normal) is consistent with the results of Sellier (1987), Cheah *et al.* (1995) and Leach *et al.* (1996), which indicate that crossbred Nn genotypes (Landrace x Large White) show a large variation in meat quality (as defined by pH_i), with 43% of the pigs normal and 57% with PSE meat. The halothane gene, in a recessive homozygous form, will initiate the onset of malignant hyperthermia (MH) when the live genotypes are exposed to stress stimuli (Mitchell & Heffron, 1982). This probably explains the low mean pH_{45} value of the nn genotypes, suggesting that certain preslaughter factors (transport, handling, stunning) acted as a trigger mechanism in precipitating the rapid and extensive post mortem glycolysis. Kaufmann *et al.* (1993) concluded that a reliable assessment of meat quality can only be done after full rigor development and that the initial pH measurements for determining meat quality should be used when groups of carcasses, rather than single carcasses, are measured. Inclusion of the final pH in meat quality assessment would also identify carcasses with late developing PSE (Hampshire effect), due to a normal initial rate but extended duration of glycolysis, such as was experienced with some of the NN genotypes, of which 36% of the carcasses were identified with a pH_{24} below 5.5. As the mean pH_{24} value for the nn genotypes was below 5.5, which is the isoelectric point of the principal muscle proteins (Lawrie, 1984), the subsequent denaturation of these proteins were acute, thus explaining the high drip losses incurred (Table 10.6.1.1). This is confirmed by correlation values (Table 1, Annex A) showing a significant ($P < 0.05$) negative correlation between pH_{45} and drip loss (r = -0.77). However, the correlation between pH_{24} and drip loss was not as strong (r = -0.29), which is probably because the difference between genotypes for ultimate pH (pH_{24}) was not as much as that of the initial pH (pH_{45}). The ultimate pH (pH_{24}) is determined by muscle glycogen potential (sum of the concentration of glycogen, glucose-6-phosphate, glucose and lactic acid) prior to slaughtering

(Monin *et al.*, 1981), whereas the rate of pH decline post mortem is determined by, among others, halothane genotype (Monin & Sellier, 1985).

The use of on-line pH measurement as a means to determine meat quality has been the subject of various discussions (Warriss & Brown, 1987; Bendall & Swatland, 1988; Swatland, 1995). Various objective techniques have been evaluated, however, several questions remain unresolved, such as time of measuring, anatomical measuring position and equipment (Lündstrom *et al.*, 1987). The South African pork industry does not currently have any nationally accepted system whereby any discrimination on the basis of meat quality is made at the point of slaughter or otherwise. A number of South African stud breeders and producers discriminate against the halothane gene in their breed animal selection programs, but this alone will not eradicate PSE meat. A well designed meat quality evaluation system should be able to give much needed additional information on environmental factors which do cause PSE meat (regardless of genotype) and thus assist in addressing this problem. The international meat industry has undergone significant improvements in both eliminating the halothane gene through genetic selection of breed stock and lowering the incidence of PSE meat by increasing line speeds and refrigeration (Swatland, 1995). These improvements may have weakened the predictive value of on-line pH measurements, but future pH measurements may involve superior methods such as neural networks coupled with other on-line meat quality measurements.

Table 10.6.1.1 The effect of genotype and sex on meat quality characteristics (least square mean ± std. error)

	Genotype			Sex		Gen x Sex interaction
	NN	Nn	nn	Barrow	Gilt	P > t
Chilling loss (%)	2.25 ± 0.253	2.49± 0.289	1.95 ± 0.316	2.19 ± 0.269	2.23 ± 0.219	0.642
Drip loss (%)	1.53^{a} ± 0.148	2.30^{b} ± 0.199	3.67^{c} ± 0.248	2.63 ± 0.234	2.41± 0.191	0.975
pH_{45}	6.22^{d} ± 0.051	5.94^{e} ± 0.061	5.36^{f} ± 0.066	5.84 ± 0.053	5.84 ± 0.043	0.931
pH_{24}	5.62^{a} ± 0.032	5.60^{a} ± 0.038	5.44^{b}± 0.041	5.58 ± 0.033	5.53 ± 0.027	0.283
L*	42.0^{a} ± 0.474	43.5^{b} ± 0.563	45.6^{c} ± 0.611	43.7 ± 0.495	43.7 ± 0.403	0.301
a*	5.51 ± 0.186	5.40 ± 0.221	5.81 ± 0.240	5.44 ± 0.194	5.71 ± 0.158	0.589
b*	6.74 ± 0.202	6.67 ± 0.240	7.20 ± 0.260	6.77 ± 0.211	6.98 ± 0.172	0.090
Cooking loss (%)	25.6^{d} ± 0.281	26.4^{d} ± 0.335	28.2^{e} ± 0.363	26.8 ± 0.294	26.7 ± 0.239	0.495
Warner-Bratzler (kg/1.27cm)	2.92^{d} ± 0.076	3.35^{e} ± 0.090	3.11^{d} ± 0.098	3.14 ± 0.079	3.10 ± 0.066	0.054
Moisture (%)	71.8^{a} ± 0.245	73.0^{b} ± 0.292	71.8^{a} ± 0.317	72.3 ± 0.256	72.1 ± 0.209	0.088
Fat (%)	2.02 ± 0.186	1.84 ± 0.221	1.76 ± 0.240	1.78 ± 0.194	1.97 ± 0.158	0.591
Protein (%)	21.9^{a} ± 0.190	21.0^{b} ± 0.225	22.3^{a} ± 0.245	21.7 ± 0.198	21.7 ± 0.161	0.058
Ash (%)	1.38 ± 0.067	1.33 ± 0.080	1.47 ± 0.086	1.29^{a} ± 0.070	1.50^{b} ± 0.057	0.798

[a-c] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.05$), according to contrast analyses (see text).
[d-f] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.001$), according to contrast analyses (see text).

10.6.2 Chilling and drip loss

The mean chilling loss values (the difference in weight between the warm and cold carcass as a percentage of warm carcass weight) were the highest (2.49%) for the carcasses from the heterozygous (Nn) genotypes, with NN being intermediate (2.25%) and nn the lowest (1.95%), although comparison of the genotypes (Table 10.6.1.1) did not reveal significant differences ($P > 0.05$). Drip loss values did show considerable statistical differences ($P < 0.05$) between the halothane genotypes, with the nn genotypes having the highest value (3.67%) and NN the lowest value (1.53%). The absence of genotype x sex interaction and sex differences indicate that this financially important characteristic is directly due to the presence of the halothane gene.

The high drip loss (3.67%) observed in the nn genotypes can be ascribed to numerous causes, such as protein denaturation, a rapid pH decline and a low initial pH. Numerous other studies showed that in the presence of the halothane gene fluid loss is increased (Lündstrom *et al.*, 1989; Murray *et al.*, 1989; Sather *et al.*, 1991a,b; De Smet *et al.*, 1992), especially when the gene is present in the homozygous (nn) form. The lack of statistically significant differences between genotypes for chilling loss might be ascribed to the fact that the carcasses were still intact when these measurements were made, thus trapping fluid in areas such as within and between undamaged muscle fibres. Although fluid may have been released from the filament lattice and transferred to the interfibre space (Swatland, 1995), it was not lost from the intact carcass. The lower chilling loss value (1.95%) of the nn genotypes may also be due to an increased carcass chilling rate, since the insulating subcutaneous fat layers were generally thinner (Table 10.5.1.1) compared to the other genotypes. However, cutting across the muscle fibres, as is done when determining drip loss, could result in this trapped fluid being released, causing a more severe weight loss from the carcass as was shown by Smith and Lesser (1979), who indicated that cutting PSE pork increased drip loss from 0.77% to 1.70% of trimmed carcass weight. However, somewhere in the processing line muscle will be cut with a resultant drip loss. The method used in this investigation therefore gives a more accurate indication of real drip loss as experienced in the processing line.

The results from Table 10.6.1.1 show that the nn genotypes have pH values (pH_{45} and pH_{24}) close to 5.5, which thus causes increased drip loss. Water holding capacity (WHC), measured as drip loss, generally decreases when the pH is low or the rate of pH decline is rapid (Swatland, 1995). This situation is exacerbated if accompanied by a high internal carcass temperature, leading to protein denaturation and membrane leakage, a direct cause of increased drip loss (Honikel, 1985; Honikel & Kim, 1986). The pattern of drip loss among the genotypes suggest that the presence of the gene,

either in the Nn or nn genotypes, will lead to a lower WHC. Results from Penny (1969 and Swatland (1992) indicate that filament spacing, and thus WHC, does not decrease until the pH drops below 6.2. This seems to be consistent with the data from this investigation, however these type of results have been criticised by other meat scientists (Swatland, 1992). This is probably due to the results on drip loss (using the bag drip method such as in this investigation) from other studies showing an increase in drip loss from pH 7 to 6.1, then little further increase in moisture loss below 6.1, whereas fibrillar WHC (determined with centrifugation, where moisture is separated from the meat sample by using centrifugal forces) shows the opposite pattern, with drip loss increasing rapidly at lower pH values.

The larger lean to fat ratio and the increased eye muscle area (Table 10.5.1.1) of the sample from genotypes with the halothane gene (Nn and nn) used for drip loss determination may also contribute to an increase in drip loss, since more lean is exposed to the atmosphere and could contribute to more fluid escaping from between the fibres. To compare this source of variation further research is needed to compare similar cubes, rather than standardized thickness of slices (Honikel, 1998). This will compensate for eye muscle area relative to weight, but calculating the required area may be problematic.

10.6.3 Meat colour

The tristimulus (L*, a*, b*) colour evaluation (Table 10.6.1.1) of the fresh eye muscle clearly show that the meat originating from the nn genotypes have the highest mean reflectance value (L* =45.8), thus indicating paler meat compared to both the Nn (L* = 43.5) and NN genotypes (L* = 41.9). The differences between all the genotypes were significant ($P < 0.05$), with no genotype x sex interaction being noted. Comparison of the sexes (barrows vs. gilts) did not show any significant differences ($P > 0.05$) for reflectance or colour. Examining genotypes, the mean a* and b* values showed an increase ($P > 0.05$) from the NN to the nn genotypes, with the carriers (Nn) having the lowest mean values of the three genotypes. The higher a* values (higher a* values indicate an increase in redness) of the nn genotypes are probably due to moisture being lost (drip loss) and thereby increasing the concentration of muscle pigment (Ahmed *et al.*, 1990). The L* (reflectance) values are in accordance with previous studies (Leach *et al.*, 1996; Murray *et al.*, 1989), confirming the increase in light scattering associated with PSE meat (Bendall, 1973), which is due to denaturation of the sarcoplasmic proteins. Factors that contribute to the variation in opacity in raw meat are chilling rate, rate of pH decline and the available substrate (glycogen) for the conversion to lactic acid. Van der Wal *et al.* (1987) also indicated that meat colour is not

constant along the MLT, with the cranial part of the muscle being less susceptible to aberrant meat quality than the lumbar region. In accordance with the consensus of previous research, the results of this investigation for pH (pH_{45} and pH_{24}) suggests that the muscle of the nn genotype had a much higher glycolytic rate than the NN and Nn genotypes during the period immediately after slaughter, and thereby contributing to PSE meat.

As this investigation shows, the presence of the halothane gene is associated with an increase in reflectance, which is perceived by the observer as increased paleness. Since a large portion of pork is sold as fresh meat, such deviations in quality could increase consumer resistance in buying and consuming pork. Although the South African pork industry is aware of the problems associated with PSE pork, there is currently no classification system in place that discriminates against poor meat quality. Correlation values calculated in this investigation (Table 1, Annex A) indicate a significant ($P < 0.05$) negative correlation ($r = -0.67$) between pH_{45} and L* values for fresh meat, suggesting that selection of carcasses with high pH_{45} values could mean a measurable decrease in the incidence of PSE meat.

10.6.4 Cooking loss and meat tenderness

Mean cooking loss (Table 10.6.1.1) was the highest for the meat originating from the nn genotypes (28.2%), differing significantly ($P < 0.001$) from the NN (25.6%) and Nn genotypes (26.4%). Results from De Smet *et al.* (1992) indicated a similar pattern, but with higher losses. Warner-Bratzler shear values indicate that the Nn genotypes have the highest mean value (3.35 kg/1.27 cm) with nn intermediate (3.11 kg/1.27 cm) and NN the lowest (2.92 kg/1.27 cm). Only the differences between NN and Nn genotypes was significant ($P < 0.001$). Murray *et al.* (1989) reported similar results, suggesting that the halothane gene is associated with higher shear values. Kemp *et al.* (1976) reported higher tenderness in PSE pork, whereas others found better tenderness in normal meat (Buchter & Zeuthen, 1971). Tornberg *et al.* (1992) concluded that as tenderness is best correlated to the sarcomere length of raw meat, tender pork could be obtained if the meat has a low degree of contraction. Although statistically significant, the differences between genotypes are small and it remains doubtful whether the above mentioned characteristics could be detected and have a major influence on customer preferences. There were no significant sex differences and no genotype x sex interaction present for both cooking loss and shear values.

10.6.5 Chemical composition

The meat originating from the Nn genotypes had the highest moisture (73.0%) and lowest protein (21.0%) content, differing significantly ($P < 0.05$) from the NN (71.8% and 21.9%, respectively) and nn (71.8% and 22.3%, respectively) genotypes (Table 10.6.1.1). Differences between NN and nn were insignificant ($P > 0.05$). Mean muscle fat (NN = 2.02%, Nn = 1.84% and nn = 1.76%) and mean ash (NN = 1.38%, Nn = 1.33% and nn = 1.47%) values showed insignificant differences between genotypes. None of the above mentioned chemical characteristics showed genotype x sex interaction and differences between sexes were not significant. The results of this investigation is partially in accordance with Murray *et al.* (1989) and Jones *et al.* (1988), who reported that the presence of the halothane gene is accompanied by an increase in protein and lower intermuscular fat, with the Nn genotypes having intermediate values for fat and protein. Differences in chemical composition were small, although statistically significant, in % moisture and % protein. These differences are difficult to explain. For example, it is known that protein content is highly correlated with moisture content (Murray *et al.*, 1989), but the present results do not support this (Table 10.6.1.1). On the other hand, other studies indicated a lower intramuscular fat content in muscles from nn genotypes (Jones *et al.*, 1988), whilst only a tendency could be observed in the present investigation.

10.6.6 Conclusion

A general overview of the results presented in Table 10.5.1.1 (carcass quality characteristics) and Table 10.6.1.1 (meat quality characteristics) show that the presence of the halothane gene, especially in the nn genotype does have several advantages with regard to carcass composition (high lean yield, low fat/skin content) compared to the NN genotype. With the current South African classification system these genotypes do have a distinct advantage in that the producer is paid for the higher lean yield, without any penalties for poor meat quality. However, the meat quality characteristics of the nn genotypes indicate poor fresh meat quality with regard to low initial pH values (with a resultant increase in drip loss) and an increase in paleness (high L* values), which results in an inferior product offered to the consumer. The results would suggest that the Nn genotype (which consistently has intermediate results for both carcass and meat quality) offers the best compromise in terms of good carcass quality and acceptable meat quality. However, the gains in carcass lean yields are generally small and it is doubtful if the producer would reap the financial benefit since the current classification system could not distinguish between NN and Nn genotypes for LMP in this investigation. Most of the meat quality characteristics (pH_{45}, drip loss, L* values) are intermediate but the mean pH_{45} values would suggest borderline PSE meat, which would not be

acceptable as good quality fresh meat. These results therefore suggest that in order to ensure a consistent and an acceptable fresh pork meat quality, the use of the halothane gene is not recommended:

10.7 PROCESSED PRODUCTS

Four types of processed (back bacon, canned hams, fresh sausage and viennas) products were manufactured from the meat derived of each of the three genotypes. The meat used for the canned hams, fresh sausage and viennas were pooled within genotype, therefore data were analysed with genotype as the only factor. The data for back bacon were analysed using the full model, with both genotype and sex as factors.

10.7.1 Back bacon

The present investigation was designed to investigate the effect of genotype (and not necessarily meat type, that is normal, PSE and DFD meat) on pork processing characteristics. Therefore statistical analysis could not be performed on meat type, but only on genotype and sex. Since very little literature is available on processing characteristics of genotypes, comparison to meat types is considered appropriate, and are presented throughout the text. The results in Table 10.7.1.1 are expressed as losses or gains in weight of the defrosted backs prior to any processing. Examining Table 10.7.1.1, it is clear that the genotype x sex interaction was absent for all the variables measured, and that values for sexes did not differ statistically.

Moisture loss during thawing (thaw loss) did not differ ($P > 0.05$) among genotypes (Table 10.7.1.1), however the initial weight gain (pumped gain) during brine injection was almost double for the backs originating from the NN (15.2%) genotype, with nn having the least (8.9%) gain ($P < 0.001$). Fluid loss after tank curing (brine loss) did not differ among genotypes. Bearing in mind that all nn genotypes resulted in PSE meat, the current results support various reports (Wismer-Pedersen, 1960; Wismer-Pedersen, 1968; Smith & Lesser, 1982) indicating that PSE meat generally absorbed less brine, and thus less salt, than normal meat. The accompanying low WHC typical of PSE meat was probably due to extensive protein damage incurred during the rapid post mortem glycolysis. This protein denaturation offset any advantage in WHC expected due to the more open structure and a consequently greater formation of a salt/protein complex. This is further supported by the current results with a significant ($P < 0.05$) reduction in total gain for the nn genotypes, compared to the NN and Nn genotypes. Colour and reflectance values (CIELAB) did not differ significantly between any back bacon samples originating from the three genotypes (Table 10.7.1.1). This result is difficult to explain, since all the backs originating from the nn genotype were of the PSE type. One would therefore expect these processed products to be paler in colour as well.

The predominant muscle in the bacon manufactured for this investigation was the MLT, a muscle classified as white, predominantly anaerobic and prone to the development of PSE. This was done in an effort to highlight the characteristics of PSE meat during the manufacturing process. A low pH is preferable in preparing cured products since it enhances weight gain during curing as well as reducing shrinkage during maturation (Wismer-Pedersen, 1959b). However, the low pH levels associated with PSE meat (and the halothane gene) and the subsequent reduction in net product yield for back bacon, which is regarded as an important value added pork product in South Africa, should discourage both producers and processors using the halothane gene commercially, since it could produce processed products with lower yields and thus lower financial returns.

Table 10.7.1.1 The effect of genotype and sex on back bacon quality characteristics (least square mean ± std. error)

	Genotype			Sex		Gen x Sex interaction
	NN	Nn	nn	Barrow	Gilt	P > t
Thaw loss (%)	6.07 ± 1.09	7.12 ± 1.30	5.08 ± 1.41	6.93 ± 1.14	5.25 ± 0.927	0.287
Pumped gain (%)	15.2^{d} ± 1.00	14.9^{d} ± 1.18	8.9^{e} ± 1.29	14.2 ± 1.05	11.8 ± 0.852	0.459
Brine loss (%)	5.21 ± 1.13	3.46 ± 1.34	5.54 ± 1.45	5.14 ± 1.18	4.33 ± 0.957	0.752
Total gain (%)	10.0^{a} ± 1.46	11.5^{a} ± 1.74	3.4^{b} ± 1.88	9.09 ± 1.53	7.48 ± 1.24	0.378
L*	38.6 ± 0.316	39.0 ± 0.376	39.6 ± 0.408	39.3 ± 0.330	38.7 ± 0.269	0.731
a*	4.88 ± 0.104	4.84 ± 0.123	5.11 ± 0.134	4.94 ± 0.108	4.94 ± 0.088	0.853
b*	2.36 ± 0.172	2.36 ± 0.205	2.72 ± 0.222	2.63 ± 0.180	2.34 ± 0.146	0.923

[a-c] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.05$), according to contrast analyses (see text).
[d-f] Values in the same row with the same caption (genotype or sex) with different superscripts differ ($P < 0.001$), according to contrast analyses (see text).

10.7.2 Canned Hams

Both the untreated and phosphate (sodium tripolyphosphate) treated canned hams (Figure 10.7.2.1 and 10.7.2.2) made from meat originating from the NN genotype had significantly less ($P < 0.001$) unbound moisture (measured as % cooking loss) after sterilization than the nn derived hams (Table 10.7.2.1). The hams made from meat originating from the heterozygotes (Nn) also had significantly higher ($P < 0.001$) cooking losses compared to the NN hams, but did not differ from the nn hams. In the case of the hams manufactured with phosphate, the hams originating from the Nn genotype had a higher ($P < 0.001$) cooking loss (16.87%) compared to the NN hams (13.75%) and nn hams (17.73%), but differences between Nn and nn were insignificant. Results were similar for the untreated hams (NN = 27.92%, Nn = 30.12% and nn = 31.14%).

Table 10.7.2.1 Mean values (with standard errors) of cooking loss (%) in canned hams, treated with and without phosphate (n = 10 cans/genotype)

	Genotype		
Characteristic	NN	Nn	nn
No phosphate	$27.92^{d} \pm 0.423$	$30.12^{e} \pm 0.423$	$31.14^{e} \pm 0.423$
Phosphate	$13.75^{d} \pm 0.731$	$16.87^{e} \pm 0.731$	$17.73^{e} \pm 0.731$

d-f Values in the same row with different superscripts differ ($P < 0.001$), according to contrast analyses (see text).

Fisher and Mellett (1997) reported similar cooking loss results for canned hams made with phosphates (0.9%), indicating significantly higher cooking losses for hams made from meat originating from Nn and nn genotypes, compared to that from the NN genotype (NN = 13.33%, Nn = 16.73% and nn = 15.54%). In this investigation the addition of 0.3% polyphosphates to the ham mixture lowers the cooking losses by approximately 50% in all three genotypes, but still fails to neutralise the effect of the halothane gene with respect to higher cooking losses. The pH values (pH_{45} and pH_{24}) measured in this investigation showed that the NN genotypes had a much higher initial pH (pH_{45} = 6.22) than the Nn (pH_{45} = 5.94) and nn genotypes (pH_{45} = 5.36), suggesting reduced WHC for both Nn and nn genotypes. The reduced WHC of PSE meat leads to increased cooking losses in the cans due to a higher than normal degree of aggregation of meat proteins, especially for pasteurized, canned hams (Wismer-Pedersen, 1968). PSE hams processed with polyphosphates showed significant improvement in WHC and technological yield (Davis *et al.*, 1975b). Siedlecki (1965, as cited by Ellinger, 1972) reported that the addition of 0.35% polyphosphates to canned hams reduced the free juices by 5% and improved product firmness.

Comparisons to non-canned products, such as whole cured hams, also help to illustrate the processing problems associated with PSE meat. Honkavaara (1988), comparing PSE pork

($pH_1 < 5.8$) with non-PSE pork ($5.8 < pH_1 < 6.4$) in cooked cured ham production, reported that PSE pork resulted in acceptable yet decreased sensory scores and a technological yield of 94.0 %, compared to 105.9 % for non-PSE ham. Similar relationships between pH and ham quality was reported by Müller (1991), indicating that increased pH values resulted in higher ham yields, with a concomitant decrease in the amount of juice exudation. The hams with the higher pH levels were also more tender compared to hams made from PSE meat.

In this investigation the tumbling period used for each ham mixture was short (25 min continuously), thus enhancing the differences in cooking losses observed between the genotypes. Ockerman *et al.* (1978) reported that short term tumbling (30 min) improved muscle cohesion but did not significantly affect yield. Apparently a longer time is required to increase yield when compared to cohesion. Increased massaging also resulted in an increased distribution of cure ingredients with a resultant more uniform colour (Gillett *et al.*, 1981). Krause *et al.* (1978) and Ockerman *et al.* (1978) reported similar results for tumbling.

As the hams are contained in sealed cans the processor does not suffer any direct economic consequences, since all the moisture is retained in the can and not lost during processing. However, the quality of the hams are compromised by factors such as increased gel cookout on the surface and non uniform colour. The addition of phosphates can correct some of the effects of PSE meat, however, food and health legislation (depending on the country) should be observed if phosphates are used. These results emphasises the need for stringent quality control over meat used in processing, such as an on-line meat quality evaluation system based on initial pH measurements, that would identify meat not suitable (i.e. PSE meat) for certain processed products like back bacon or canned hams.

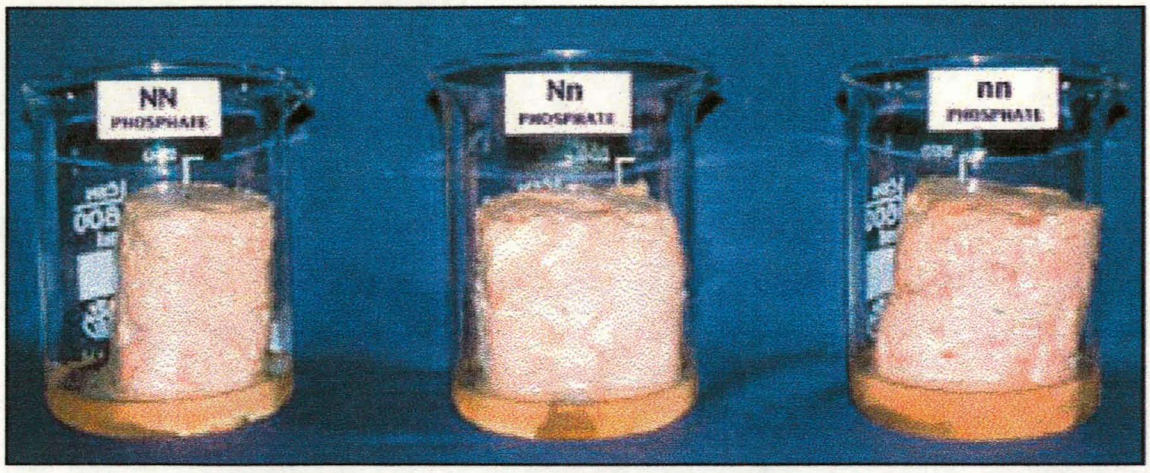

Figure 10.7.2.1 Canned hams derived from the three different genotypes, with phosphate

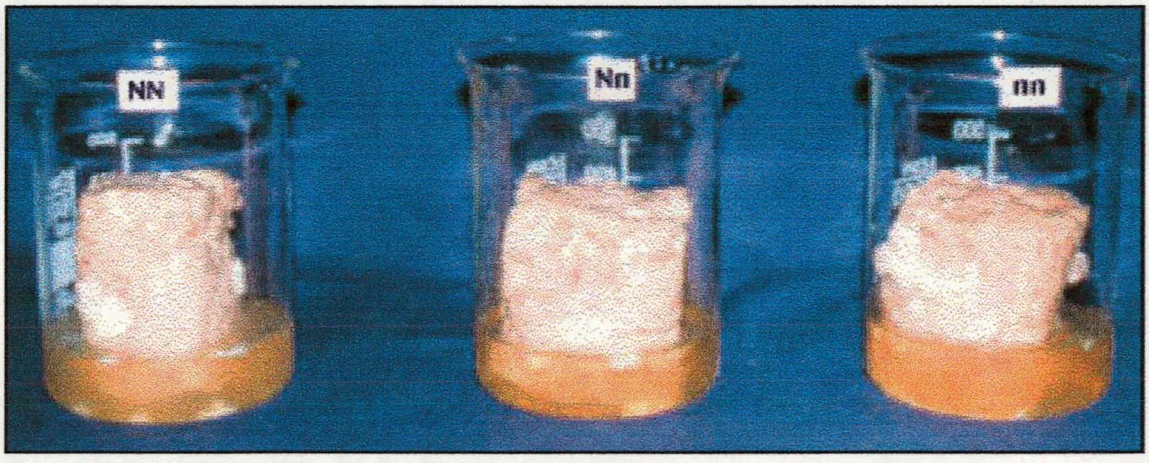

Figure 10.7.2.2 Canned hams derived from the three genotypes, without phosphate

10.7.3 Fresh pork sausage

10.7.3.1 Processing losses of fresh pork sausages

The fresh sausages made from the meat originating from the three halothane genotypes were subjected to two treatments (rusk added and no rusk added) to determine if the addition of a moisture retaining agent (rusk) would reduce the moisture loss of low ultimate pH meat, such as that associated with the halothane gene. No phosphates were added to either treatment. Phosphates act primarily as moisture retaining agents and would therefore further confound the results. The genotypes were compared within each treatment to determine the effect of the presence of the gene on WHC. The meat used for the preparation of the fresh sausage consisted of the topside (*M. biceps femoris, M gracilis*, the adductors of the thigh and part of the *M. quadriceps*) and silverside (*M. semimembranosus*), both cuts classified as white fast glycolysing muscle groups.

The sausage manufactured without rusk had the highest chilling loss for the nn genotype, differing significantly ($P < 0.05$) from the Nn genotype, with NN intermediate. The treatment with rusk added resulted in the NN genotype having the lowest chilling loss, with Nn the highest ($P < 0.05$) and nn being intermediate. Cooking loss was significantly higher ($P < 0.05$) for the nn genotype (with no rusk added) compared to the NN genotype, with Nn intermediate (Table 10.7.3.1.1).

Table 10.7.3.1.1 Chilling and cooking losses of the two types of fresh sausage.

Characteristic	Genotype NN	Nn	nn
No rusk			
chilling loss (%)	$3.77^{a,b} \pm 0.215$	$3.15^{a} \pm 0.215$	$3.82^{b} \pm 0.215$
cooking loss (%)	$11.93^{a} \pm 0.887$	$14.23^{a,b} \pm 0.887$	$15.07^{b} \pm 0.887$
total loss (%)	$15.7^{a} \pm 0.937$	$17.4^{a,b} \pm 0.937$	$18.9^{b} \pm 0.937$
Rusk			
chilling loss (%)	$3.05^{a} \pm 0.213$	$3.69^{b} \pm 0.213$	$3.42^{a,b} \pm 0.213$
cooking loss (%)	9.10 ± 0.905	9.32 ± 0.905	10.74 ± 0.905
total loss (%)	12.6 ± 0.904	13.0 ± 0.903	14.2 ± 0.904

$^{a-c}$ Values in the same row with different superscripts differ ($P < 0.05$), according to contrast analyses (see text)

The results for chilling and cooking losses were unexpected, with the Nn sausages showing both the highest (with rusk addition) and lowest chilling losses (without rusk). It was therefore decided to examine the total processing and cooking losses to explain these results. Without rusk addition,

significant differences between genotypes could be demonstrated, with nn losing the most (18.9%) and NN the least (15.7%) moisture ($P < 0.05$). However, the sausages with rusk addition showed no significant differences between genotypes, but these results do suggest an increase in total moisture loss with inclusion of the halothane gene.

This lack of clear differences between genotypes could be due to factors such as moisture losses prior to manufacturing, type of sausage casing, addition of salt, cooking time and severity of the grinding process. Since no fat was added to the sausage mixtures, fat content could not have had a significant effect on the differences in cooking losses. This is supported by results from Berry and Leddy (1984) indicated that pan frying of ground beef with increasing levels of fat does not increase cooking losses. Regardless of ultimate pH level, the genotypes with the halothane gene (Nn, nn) still had a tendency to produce higher levels of exudation during storage and cooking, though not always statistically significant. The inclusion of salts (e.g. sodium chloride) which enhances WHC regardless of pH level (Hamm, 1960), could have influenced the results of this investigation. However, levels of salt inclusion were similar for both treatments and genotypes. Internal temperatures during cooking were in the range of 73 - 78°C, thus a loss in WHC due to muscle protein denaturation, other than that caused by grinding, is also expected (Lawrie, 1991). The cooking time was short (app. 12 min), thereby possibly limiting larger differences in moisture loss between genotypes. These results would suggest that PSE pork can be used with a certain amount of success for certain products (such as fresh sausage) which undergo dramatic changes in textural structure through processes such as grinding. Partly due to the use of moisture binding agents and type of sausage casing, moisture losses (both chilling and cooking loss) were kept comparatively (between genotypes) low and would thus indicate a viable use for lower quality (PSE) pork in selected value-added products.

10.7.3.2 Sensory evaluation of fresh pork sausages

The fresh sausages were evaluated for juiciness comparing genotypes within each treatment (rusk and no rusk). Five descriptive classes were used (very dry, dry, not dry/not juicy, juicy and very juicy) and each panelist was required to indicate which word was the most descriptive of each cooked sample's characteristic with regard to juiciness. Subsequently, the frequency for each class was calculated and presented in bar charts (Fig. 10.7.3.2.1. and Fig. 10.7.3.2.2)

Writing μ_{NN} for the mean juiciness of the sausage prepared from the meat of the dominant homozygote and μ_{nn} for the corresponding mean for the recessive homozygote the contrast $\mu_{NN} - \mu_{nn}$ was estimated to be 0.0426 ± 0.1219 in the case of the sausage with no rusk added, this corresponds to an approximate significance level (SL) of 0.7266 for the null hypothesis $\mu_{NN} - \mu_{nn} = 0$. The null hypothesis for the second contrast, $\mu_{Nn} - (\mu_{NN} + \mu_{nn})/2 = 0$ had a SL = 0.2698, corresponding to an estimate of 0.0780 ± 0.0706. In the case of the sausage with rusk the estimates were 0.3110 ± 0.1253 (SL = 0.0131) and 0.0222 ± 0.0718 (SL = 0.7571), respectively.

In the sausages prepared with no rusk the evidence suggests that the mean juiciness was the same for the three genotypes. In the case of the sausage prepared with rusk there was strong evidence to suggest that $\mu_{NN} = \mu_{nn}$ (SL = 0.0131) but there was weak evidence to contradict $\mu_{Nn} - (\mu_{NN} + \mu_{nn})/2 = 0$ (SL = 0.7571), suggesting that the juiciness of the heterozygote was neatly positioned between the juiciness of the heterozygotes at the extremes. Sausage (with rusk) prepared from meat originating from the nn genotype was juicier than the sausage derived from the other genotypes.

These results seem to suggest that the inferior WHC associated with the halothane gene resulted, in the case of the sausage with rusk added, in a more juicy sausage since the rusk bound most of the moisture during manufacturing. This is partially supported by the results for the sausages made with no rusk, where the taste panel could distinguish between the three genotypes. The type of sausage casing used (collagen type) could also have contributed to a higher moisture content, since it is less permeable and thus more effective in retaining moisture in the test sample.

The subjective colour evaluation of the two types of fresh sausages indicate that the sausages made from the meat of the nn genotype have a darker pink colour than both the sausages made from the meat of the Nn and NN genotypes. This result seems to be consistent for both treatments (Figure 10.7.3.1 and 10.7.3.2), suggesting that the higher moisture content of the sausages made from the meat of the NN and Nn genotypes could have diluted the myoglobin concentration, resulting in a lighter red colour (Ahmed *et al.*, 1990). Do note that the x-axis in Figures 10.7.3.1 and 10.7.3.2 do

not represent any numerical values (Although equal distances between the classes dark pink, pink and light pink are used to illustrate the frequency distributions, equal distances between these classes are not implied).

These results are contrary to the common perceptions that PSE pork should be pale, even in a manufactured product. If higher moisture losses from PSE meat can be contained by moisture binding agents, such as rusk, it does leave the processor some options other than selling such meat at reduced prices or for use in lower priced products such as emulsions. Nevertheless, this should not encourage the use of the halothane gene in any genotype (Nn or nn), since the results from this investigation suggest that most manufactured pork products will suffer, in varying degrees, from reduced quality characteristics (colour, drip loss) if PSE meat is used.

10.7.4 Viennas

Compared to the differences in processing losses for the other products (Table 10.7.1.1, 10.7.2.1 and 10.7.3.1), the differences in smoking and cooking loss, for viennas, between genotypes were small (Table 10.7.4.1), suggesting that products processed to a higher degree are less susceptible to deviations cause by poor meat quality. Smoking and cooking losses were the highest for the viennas prepared from the meat originating from the nn genotype, whilst the Nn genotype had the lowest loss ($P < 0.05$). The NN genotype gave intermediate results which did not differ statistically from the other two genotypes (Table 10.7.4.1).

Table 10.7.4.1 Chilling and cooking loss (%) of an emulsion type sausage.

	Genotype		
Characteristic	NN	Nn	nn
Smoking/cooking loss (%)	$12.35^{a,b} \pm 0.378$	$11.31^{a} \pm 0.378$	$12.47^{b} \pm 0.378$

a-c Values in the same row with different superscripts differ ($P < 0.05$), according to contrast analyses (see text).

The increased chopping or cutting associated with the manufacturing of emulsion type products diminishes the differences in meat quality, especially with regard to WHC. Comminution plays an important role, apart from reducing the size of the meat pieces, in extracting salt soluble meat proteins and assisting in the binding of the product (Varnam & Sutherland, 1995). Since no phosphates were added to the emulsion, it is conceivable that the chopping and associated processes resulted in enough salt soluble proteins being extracted to neutralize the moisture loss associated with PSE meat.

10.7.5 Summary

The results of this investigation not only indicated a distinct disadvantage in the use of the halothane gene on fresh meat quality, but also on selected processed products. Whole muscle products, as in this case back bacon, made from the nn genotype suffered from low final yields, compared to the NN and Nn genotypes, which will result in financial losses to the processors. The results for canned hams indicated that the addition of phosphates can partially alleviate the problem of increased cooking losses, but with a decrease in quality (excessive gelatine formation). In addition, not all countries allow the use of phosphates in certain meat products, thus limiting the use of such agents. This investigation does suggest possible uses for PSE meat, such as for fresh sausage and emulsion products (viennas). It seems that the processes involved in the manufacturing of these products largely overshadow the effects of PSE meat (drip loss, pale colour) and that the yield losses incurred can be reduced to such an extent that it would not involve increased financial

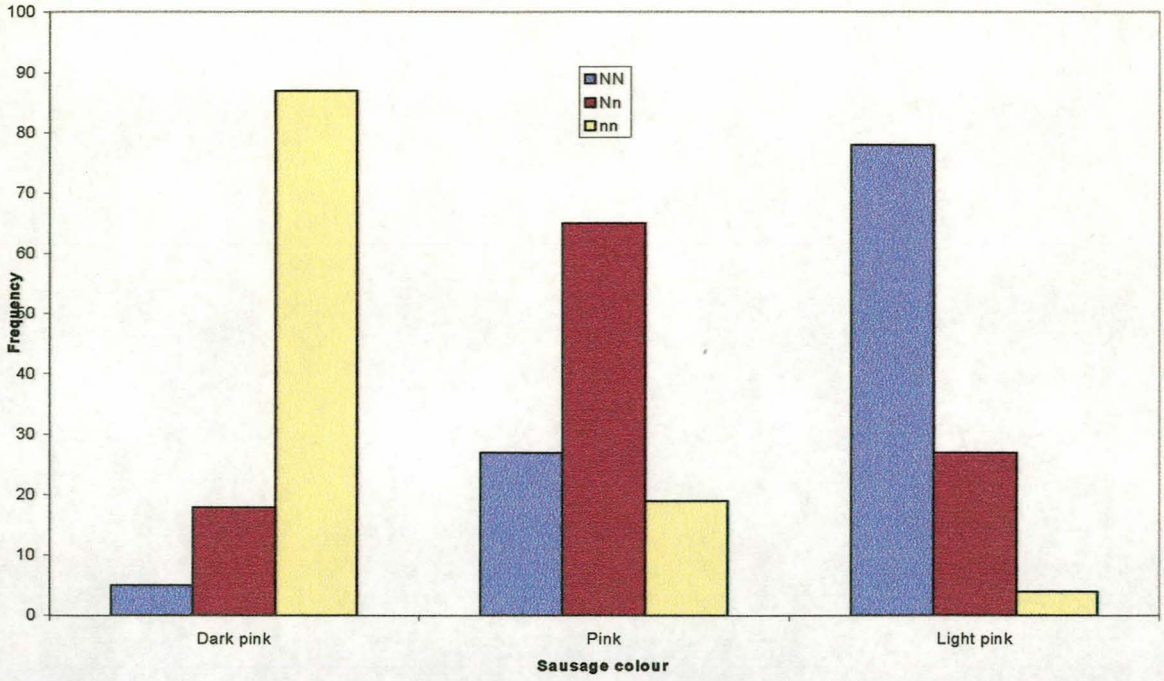

Figure 10.7.3.2.1 Colour ranking of sausages (without rusk) made from meat of different genotypes (although equal distances between the classes dark pink, pink and light pink are used to illustrate the frequency distributions, equal distances between these classes are not implied)

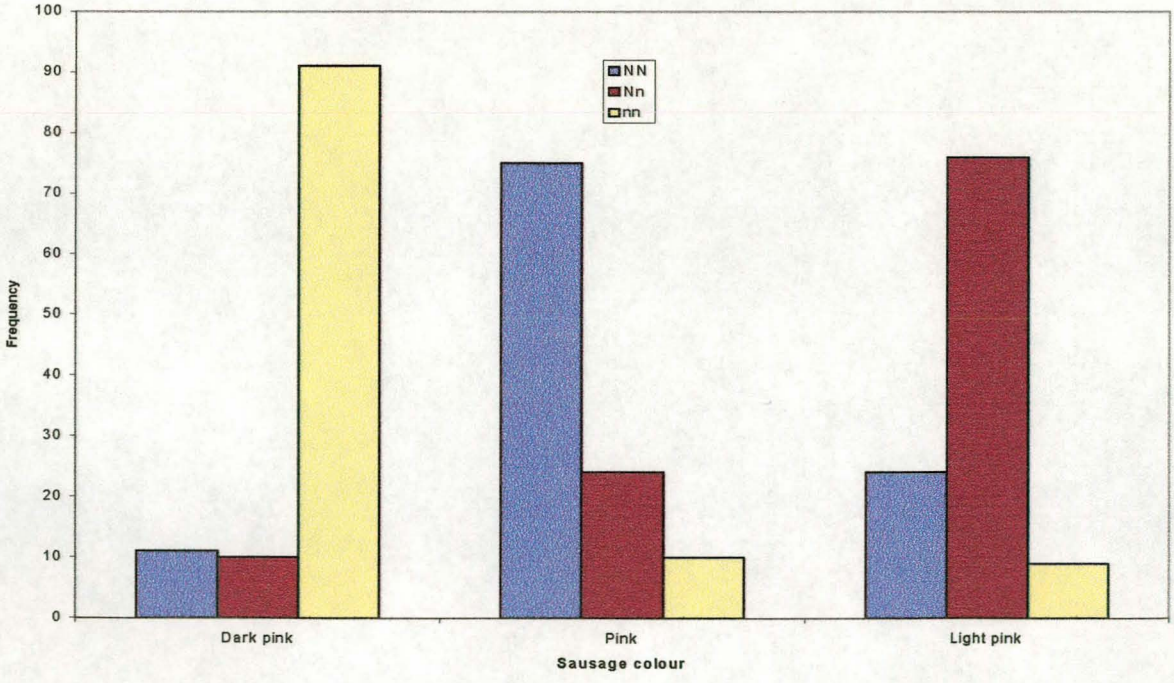

Figure 10.7.3.2.2 Colour ranking of sausages (with rusk) made from meat of different genotypes (Although equal distances between the classes dark pink, pink and light pink are used to illustrate the frequency distributions, equal distances between these classes are not implied)

losses or undue reduction in sensory qualities. However, the financial returns on these products are much lower than that obtained from selling whole muscle products.

In conclusion, the presence of the halothane gene in both the heterozygous (Nn) and homozygous (nn) form does offer advantages to the producer in terms of increased carcass lean yields, and thus increased financial returns under the present classification system e.g. LMP (nn = 69.7 % vs NN = 67.5 %, a difference of 2.2 %). However, nn carcasses loose 3.67 % vs 1.53 % (for NN) due to drip only, a difference of 2.14 %. Due to this, and assuming that most moisture will be lost from the lean portion of a carcass, the LMP advantage is slightly diminished to 67.14 % and 66.47 %, a difference of 0.64 % only. Further more, considering bacon production, a maximum gain of 3.4 % was observed for nn, while NN gained 10.0 %, a difference of 6.6 %. For the sake of illustrating the effect, and assuming that all lean meat in a carcass can be processed into bacon with similar gains as for the LMT, theoretical "processed LMP" figures of 68.46 and 73.85 % can be calculated for nn and NN, respectively, a difference of 5.39 %, but now in favour of NN. With this evidence it is clear that the disadvantages of NN in terms of lean depositioning during growth is by far over shadowed by the disadvantages of post mortem and processing losses encountered by the nn types. Meat processors can therefore pay a premium for NN genotypes, or, should the opposite be more feasible, discriminate against nn genotypes delivered to either an abattoir or processing plant.

These results should thus be seen to discourage the use of the halothane gene in production systems since the losses involved, both in terms of meat quality and financial returns, do not justify the supposedly higher returns from the increased lean content associated with the halothane gene. The design and implementation of an on-line system that can accurately detect PSE pork carcasses, as well as proper manufacturing practices to utilise PSE meat with least the deterioration in product quality (such as emulsion products), should thus become an imperative with the South African meat industry, particularly if it wishes to compete successfully in the international market.

10.8 CORRELATION AND REGRESSIONS

The correlation coefficients (r) are given in Annex A (Table 1 and 2) and the regression coefficients (R^2) in Annex B (Tables 1 - 10). Correlations that are statistically significant ($P < 0.05$) are printed in bold. The regression coefficients in Annex B are limited to the first five equations for each additional variable. Those parameters that were deemed practically applicable were used as independent variables in the regression equations.

10.8.1 Selected carcass yields: shoulder

Correlation values for shoulder lean and fat yields (Table 2, Annex A) indicate that weight (live weight [r = 0.57], warm [r = 0.58] and cold carcass weight [r = 0.59]) and length (r = 0.52) have a significantly ($P < 0.05$) positive correlation with lean content. Correlation values with fat thickness (over the *M. gluteus medius* and $T_{2\text{-}3}$–45mm) are negative ($P < 0.05$), but none reach r = -0.50. Subcutaneous fat content of the shoulder has a significantly ($P < 0.05$) positive correlation with both the medial (T_1[r = 0.54], $T_{2\text{-}3}$ [r = 0.48], GM [r = 0.60]) and lateral fat thicknesses measured ($T_{2\text{-}3}$–45mm, r = 0.55). The predicted percentage lean meat (LMP) in the carcass also has a positive ($P < 0.05$) correlation with lean in the shoulder (r = 0.36) and a negative ($P < 0.05$) correlation with fat content (r = -0.56).

The regression equations for predicting percentage fat/skin (Table 9, Annex B) in the shoulder (calculated as a percentage of cold carcass weight) suggest that meat quality indicators (pH_{45}, pH_{24}), combined with medial carcass measurements, have considerable predictive value in this investigation. The full model for percentage fat/skin in the shoulder is $R^2 = 0.60$ (10 variables), and with six variables,

$y = -7.7922 - 0.0349[CWT] + 0.00711[LNGTH] + 0.3165[pH_{45}] + 0.6761[pH_{24}] + 0.0566[T_1] + 0.0513[GM]$,

resulting in $R^2 = 0.58$ (Cp = 4.6), indicating that little additional accuracy is gained by adding more independent variables. The percentage lean in the shoulder (Table 10, Annex B) has a much lower R^2 value for the full model ($R^2 = 0.22$), supporting results from Kempster *et al.* (1982) that medial measurements taken towards the cranial end of the carcass do not have good predictive value, possibly due to the shoulder fat not being adequately supported by the vertebra, making fat thickness measurements less accurate.

Research has documented and proved conclusively that the degree of fatness, measured as fat thickness, is the most important factor affecting pork carcass yields of the four (ham, loin, shoulder and butt) primal lean cuts and that fat thickness is highly related to percentage yields of these cuts. Various reports (Kempster & Evans, 1979; Fortin *et al.*, 1981) have indicated that, generally, dorsal

midline fat measurements on split carcasses in the shoulder region have low predictive value for lean content, whereas the lumbar and posterior thoracic region show the highest predictive value. The reasons for this are not clear; it is possible that the shoulder measurements, which are furthest from the initial splitting point, is more sensitive to the accuracy with which carcasses are split. Another source of inaccuracy is that the shoulder fat is less well supported by the vertebral spines than the rump fat. Furthermore, the *M. gluteus medius* provides a clear anatomical point of reference for midline measurements (Kempster *et al.*, 1982).

10.8.2 Selected carcass yields: leg

The results from this investigation indicate significant positive correlations ($P < 0.05$) for leg fat/skin (Table 2, Annex A) with both medial (T_1 [r = 0.53], $T_{2\text{-}3}$ [r = 0.69], GM [r = 0.71]) and lateral ($T_{2\text{-}3}$–45mm [r = 0.70]) fat thickness. In addition, MLT depth at the classification ($T_{2\text{-}3}$-45mm) point has a significantly negative correlation (r = -0.44) with leg fat/skin. Similarly, percentage predicted lean yield (LMP), calculated from both the muscle and fat depth at $T_{2\text{-}3}$–45mm, is negatively correlated (r = -0.70; $P < 0.05$) with leg fat. The lean cuts in the leg (topside, silverside, thickflank and rump) are all negatively correlated with the medial and lateral fat thickness values (Table 2, Annex A). The results from this investigation indicate that the fat thickness at the classification point ($T_{2\text{-}3}$–45mm) generally has the highest ($P < 0.05$) correlation values (r = -0.59, r = -0.68, r = -0.55, r = -0.47 respectively) with the four lean cuts, with the fat thickness over the *M. gluteus medius* having the second highest values (r = -0.56, r = -0.62, r = -0.44, r = -0.39, respectively). The correlation values for leg fat with medial fat thickness values decline as the measurement positions approach the cranial end of the carcass. This decline in correlation values could be due to the sensitivity of the measurements that are furthest from the initial (caudal) splitting point (Kempster & Evans, 1979).

Regression equations for total lean yield (as a percentage of cold carcass weight) calculation in the leg show an $R^2 = 0.78$ (Cp = 4.4) when six variables (WWT, CWT, pH_{45}, pH_{24}, GM and FAT) are used:

$y = 36.9561 + 0.3212[WWT] - 0.3446[CWT] - 0.3247[pH_{45}] - 2.7712[pH_{24}] - 0.2110[FAT] - 0.0443\ [GM])$

The results in Table 4 (Annex B) indicate that warm carcass weight, fat thickness ($T_{2\text{-}3}$-45mm) and fat thickness over the *M. gluteus medius* are important variables in predicting lean content of the leg. The regression values for the four lean cuts (topside, silverside, thickflank and rump) when expressed as a percentage of cold carcass weight, indicate that both carcass weight (both warm and cold) and fat thickness (medial and lateral) have a considerable value in predicting lean cut yield. The percentage silverside (Table 6, Annex B) had a high R^2 value ($R^2 = 0.70$, Cp = 5.2) when six

variables (warm and cold carcass weight, T_{2-3} – 45mm, T_1, T_{2-3}, pH_{24}) are used:

$y = 12.4431 + 0.0784[WWT] - 0.0743[CWT] - 0.9644[pH_{24}] - 0.0568[FAT] - 0.0204[T_1] - 0.0293[T_{2-3}]$

The R^2 value for percentage topside (Table 5, Annex B) is lower ($R^2 = 0.53$, full model), with five variables (warm and cold carcass weight, T_{2-3}–45mm, T_{2-3}, pH_{24}) giving a $R^2 = 0.52$, Cp = 2.7

$y = 9.0749 + 0.1558[WWT] - 0.1471[CWT] - 0.7164[pH_{24}] - 0.0550[FAT] - 0.0266[T_{2-3}]$

Both percentage thickflank (Table 7, Annex B) and rump (Table 8, Annex B) have R^2 values (for the full model) below 0.50, suggesting weaker predictive values for the variables used. Thickflank (Table 7) has a $R^2 = 0.46$ (Cp = 2.6) with four variables (cold carcass weight, T_{2-3}–45mm, length, pH_{24}) in the equation

$y = 4.9036 - 0.0154[CWT] + 0.00306[LNGTH] - 0.4909[pH_{24}] - 0.0381[FAT]$

suggesting that the inclusion of more variables does not greatly improve the predictive value of this regression equation. Similarly, the R^2 value for percentage rump (Table 8) is low, $R^2 = 0.47$ (full model), and six variables

$y = 3.2961 + 0.1348[WWT] - 0.1533[CWT] - 0.3480[pH_{24}] - 0.0356[FAT] + 0.0159[MSCLE] + 0.0204[T_{2-3}]$

giving a $R^2 = 0.46$ (Cp = 5.7). The relatively low R^2 values calculated for three of the lean cuts in the leg (thickflank, rump and topside) are due to the choice of measurements taken on the carcass. Fat thickness at the classification point (T_{2-3}-45 mm) was the best single predictor of all four lean cuts, but the inclusion of a medial fat measurement (GM) located closest to the anatomical position of these cuts did not greatly improve R^2. This is probably due to the lower predictive value of dorsal midline fat measurements for lean contents, when compared to lateral fat measurements (Fortin *et al.*, 1984). A more judicious selection of measuring points (i.e. lateral fat thickness measurements on the specific joints) should increase the precision of prediction of lean and fat content in major sample joints.

10.8.3 Meat quality

The strong negative correlation (Table 1, Annex A) between pH_{45} and both fresh meat L* value ($r = -0.67$) and drip loss ($r = -0.77$), as well as the positive correlation ($P < 0.05$) between drip loss and fresh meat L* value ($r = 0.72$), clearly indicating a relation between colour, drip loss and pH_{45}. However, colour and WHC (indirectly measured by drip loss) are not as closely related. Therefore, colour alone is not a reliable indicator of WHC. Consequently, two new class of pork, RSE (reddish pink, soft, exudative) and RFN (reddish pink, firm, non-exudative) have been defined (Kauffman *et al.*, 1992), the others being PSE, DFD and normal. Thus, combining results from more than one objective method (pH, colour), in the form of an equation or index, could differentiate between true

PSE and other quality deviations having other causes, in addition to PSE. Regression coefficients (R^2) calculated for drip loss using pH_{45} alone resulted in a $R^2 = 0.59$ 9

$y = 16.9603 - 2.4775[pH_{45}]$

and pH_{45} and L* values combined resulted in $R^2 = 0.66$

$y = 4.5860 - 1.6981[pH_{45}] + 0.1789[L^*]$

The addition of the a* value improved predictability by only 3% ($R^2 = 0.68$). The inclusion of more variables, such as pH_{24}, did not greatly improve R^2. Several studies have evaluated and compared the available methods for evaluating pork quality. Kauffman (1991) found, in a very comprehensive evaluation of methods, muscle pH to be the only method which showed any predictive potential, and in this investigation pH_{45} was of relative importance in predicting WHC and colour of fresh pork.

10.8.4 Final bacon yield

The correlation valuesbetween final bacon yield (Annex B, Table 2) and meat quality indicators such as pH_{45} ($r = 0.26$), pH_{24} ($r = 0.23$), fat thickness ($r = 0.36$), measured at the $T_{2\text{-}3}$-45 mm position and L* ($r = -0.29$) are statistically significant ($P < 0.05$), although none of the r values exceeded 0.30. The regression coefficients are low, with $R^2 = 0.36$ for the full model. The results from this investigation seem to suggest, that although the R^2 values are low, fat thickness at the classification position ($T_{2\text{-}3}$-45 mm position) makes a significant contribution in determining final bacon yield. Results from Knight and Knipe (1996) indicated that the best prediction equation ($R^2 = 0.36$) for final loin yield (cured and cooked) included variables such as Minolta chromameter 'a' values and Sensoptic conductivity values. However, the low R^2 value for predicting final bacon yield (Table 2, Annex B) calculated in this investigation would not justify inclusion in a prediction equation for a commercial processing plant.

10.8.5 Conclusion

The results from this investigation suggest that fat thickness, both lateral and medial can, combined with other measurements (warm and cold carcass weight), explain to a high degree the variability in fat and lean yields from both the shoulder and leg. These results are supported by a number of investigations (Kempster & Evans, 1979; Kempster *et al.*, 1982) that reported similar results. In most of the regression equations (Annex B) lateral fat thickness ($T_{2\text{-}3}$-45mm) is identified as the most important variable, and in combination with medial fat thicknesses (*M. gluteus medius*, $T_{2\text{-}3}$), can explain a large proportion of the variation in lean content within the leg. The lack of a high R^2 value for shoulder lean content is probably due to the unsuitability of the fat thickness measurement (T_1) in predicting lean yield. A possible explanation for inclusion of meat quality predictors, such as pH_{45}, pH_{24} and L* values, in the regression models for fat and lean yields, could be explained by the

relation between post mortem pH levels (and associated meat quality indicators) and the genotypes with the halothane gene (Nn, nn), which tend to have a higher lean content and lower fat content in the selected cuts compared to the cuts derived from the NN genotypes.

The results from this investigation suggest that pH_{45}, in combination with reflectance (L*), can predict drip loss reasonably accurately. This emphasises the role of rapid post mortem glycolysis and its effect on moisture loss, which is one of the criteria by which meat quality is determined. The high (negative) correlation between pH_{45} (Table 1, Annex A) and reflectance (L*) also highlights the effect of a rapid decline in post mortem pH on colour, which is used to determine meat quality. This investigation indicates that the relation between pH_{45}, L* and drip loss is strong (Table 1, Annex A). Some studies (Garcia, 1992) have indicated similar strong relations between pH, light reflectance as well as electrical conductivity, whereas others (Garrido *et al.*, 1994) have indicated weaker relationships. However, the use of initial pH measurements (combined with other measurements such as reflectance) as a predictor of ultimate meat quality in industrial application is widespread (Banon *et al.*, 1997) as is supported by the results in this investigation. The application of a meat quality classification system based on the above mentioned variables (pH, reflectance) would not only identify PSE carcasses, but also assist processing plant management in making decisions on which carcasses are suitable for fresh meat or different processing applications, as well as determining the actual cost/losses involved in the use of PSE meat.

11 DEVELOPMENT OF OBJECTIVE PROCEDURES TO IDENTIFY PSE PORK AND PREDICT PROCESSED PORK QUALITY

A number of objective methods are currently available for evaluating pork quality, such as surface colour and reflectance, internal reflectance by fibre-optics and direct muscle pH. Some of these methods are being used by U.S. meat companies to sort pork into different quality classes. These more objective methods for evaluating pork quality have been developed and evaluated by comparing the results of each method to WHC, as determined by centrifugation (Swatland, 1986), the press method (Jones *et al.*, 1984) or bag drip loss (Somers *et al.*, 1985); ultimate pH (pH_u) (Swatland, 1986); pH at 45 min post mortem (pH_{45}) (Barton-Gade, 1981; Warriss & Brown, 1987); subjective colour and structure scores (Fortin & Raymond, 1987; Swatland, 1989; Warriss *et al.*, 1989); carcass weights and fatness (Chizzolini *et al.*, 1988); consumer panels (Somers *et al.*, 1985); or whether the carcasses were from halothane positive or halothane negative pigs (Eikelenboom & Nanni Costa, 1988).

In addition, researchers have worked on developing fibre optic probes as a rapid, non-destructive method to identify PSE pork (Jones *et. al.*, 1984; Somers *et al.*, 1985; Swatland, 1986; Barton-Gade & Olsen, 1987; Forten & Raymond, 1987; Warriss & Brown, 1987; Eikelenboom & Nanni Costa, 1988; Warriss *et al.*, 1989; Warner *et al.* 1992), and have also studied surface reflectance and microscopic methods for evaluating pork carcass quality. In the above mentioned studies, very little research has been done to correlate fresh pork characteristics to finished, cured and cooked product characteristics, in order to predict the functionality of the fresh pork in the production of processed products, such as ham or sausage. In addition, much of the research to date has only looked at each quality characteristic individually. Recent studies have indicated that some pork is pale, but not soft and exudative, and vice versa. Kauffman *et al.* (1992) described 5 categories of pork quality which were identified as: pale, firm and non exudative (PFN); red, soft and exudative (RSE); red, firm and non exudative (RFN); pale, soft and exudative (PSE); and dark, firm and dry (DFD). Therefore, combined results from more than one objective method, in the form of an equation or index can be used to classify PSE and other deviations such as DFD.

More recently, several studies have evaluated and compared the many available methods for evaluating pork quality. In a comprehensive evaluation of methods Kauffman (1991) found that muscle pH was the only method that showed any predictive potential, and in this case, pH_{45} was relative good in predicting WHC and colour of fresh pork. Results from an investigation by Warriss *et al.* (1989) indicated that evaluation of meat quality 45 min post mortem by means of colour measurements were inadequate and could not accurately predict final meat quality. Trout (1992) also evaluated a number of these methods and found that Minolta L and b values, Colormet L

values, and fibre-optic probe values at 45 min post mortem, were most promising in being able to separate Australian pork samples into 5 quality classes. To date, objective methods have not been developed which accurately select PSE pork, based on the true functionality of the meat during further processing (i.e., curing and cooking), and little attempt has been made to predict finished product quality from fresh pork quality characteristics.

Kauffman (1996) concluded that both light reflectance (regardless of apparatus manufacturer) and pH values were effective to classify pork into four different classes with approximately 75% accuracy. Of the two methods, light reflectance seemed to be superior, but both methods were effective in identifying extreme quality conditions (PSE, DFD). However, using light reflectance and pH in one predictive model does not additively improve the overall predictive efficiency. It is quite clear that none of the current approaches are satisfactory for separating RSE from RFN. The colour brightness and pH values of RSE and RFN classes are too similar to differentiate by any technique currently available. Prediction equations proposed to predict cooking loss, cured loss and texture of cured pork loin (Knight & Knipe, 1996) gave the best results with objective colour brightness (Hunter Colorlab or Minolta L* values), with WHC and Warner-Bratzler shear values being less accurate predictors (lower R^2 values).

In South Africa objective determination of meat quality, on a commercial level, by the above mentioned methods does not exist. The local meat packers and meat processors are well aware of the problems associated with inferior meat quality (PSE, DFD), however, they seem reluctant to address and resolve this problem. The South African producers and breeders are also aware of the possible effect of the MH gene on meat quality, and a number of them are actively involved in eradicating the gene from their breeding stock. However, eradication of the gene will only partially resolve the problem of inferior meat quality, since adverse environmental conditions (transport, preslaughter handling, lairage, stunning) will still result in unacceptable and widely varying meat quality. It is therefore of utmost importance that research be done to develop a meat classification system, based on results gathered under local conditions.

Such a system, if well implemented and monitored, could serve as a valuable tool in identifying situations or practices that will lead to a deterioration of meat quality, and provide methods for rectifying such practices. It will also identify carcasses with acceptable or good meat quality, and will enable meat packers and meat processors to pay premiums to producers. In so doing, it will provide a better product to the consumer, who ultimately has the final choice of which pork product or brand he or she will buy. The results from this investigation indicate that the construction of a predictive model is possible. However, such models need to be investigated and improved with

larger numbers of animals with wider genetic diversity than that used in the present investigation. The accuracy of the prediction equations presented in this dissertation needs to be confirmed by independent studies.

12 CONCLUSIONS

The detection and identification of the basic biochemical lesion in PSS and the subsequent discovery of the single point mutation associated with MH has assisted researchers in identifying the underlying factors involved in determining certain growth, carcass and meat quality characteristics associated with the halothane gene. The aim of this investigation was to determine certain carcass and meat quality characteristics and processing properties of the three different halothane genotypes, and, if possible, to make recommendations with regard to the use of the different genotypes that will meet the demands of pork producers, pork processors and pork consumers. Apart from the bone content in the shoulder, none of the other carcass, meat and processed meat characteristics indicated any sex x genotype interaction, this further emphasises the effect of the gene in that it can override known sex influences.

12.1 Carcass quality characteristics

The advantage of inclusion of the halothane gene in producing leaner genotypes with a higher lean content was clearly reflected in this investigation. Comparison of backfat thickness taken at the classification point (between the 2nd and 3rd last thoracic vertebrae, 45 mm off midline) resulted in significant differences ($P < 0.05$) between NN and nn genotypes (17.4 mm and 13.7 mm), with Nn being intermediate (16.4 mm). This resulted in a significantly higher ($P < 0.05$) percentage carcass lean yield for the nn genotype (69.7%), compared to the NN and Nn genotypes (67.5% and 68.1%), translating into increased financial returns with the current classification system. These trends were also reflected in midline fat measurements, as well as LMT area measurements.

Yields from the two primal cuts (shoulder and leg) indicated a similar pattern, with the nn genotype having less fat and more lean tissue compared to the NN genotype, with the Nn genotype being predominantly intermediate for the majority of the yield variables measured. Comparison of the sub primal cuts indicated a similar trend, with the presence of the halothane gene resulting in higher lean yields, especially when present in the homozygous form (nn). Regression values for determining dependent variables (e.g. lean yield, primal cut yield) only showed a reasonable accuracy for prediction when calculated for primal cut lean yields in the leg, using variables such as warm and cold carcass weight, as well as the medial and lateral fat thicknesses. The R^2 values for the different primal yields ranged from 0.50 to 0.71, however, as carcass lean yield is accurately predicted using lateral fat and muscle measurements, it is doubtful if additional measurements will increase the accuracy of predicting total lean content.

The results for carcass measurements and carcass yields in this investigation are in support of most of the published research reports and further emphasises the advantages of the halothane gene when

the focus is on producing superior (leaner) carcasses, with resultant higher financial returns. However, meat quality is severely affected by the presence of the gene, and this is clearly demonstrated in this investigation.

12.2 Meat quality characteristics

Some very important meat quality characteristics (pH_{45}, drip loss, colour, chemical composition) can be ascribed to the presence of the halothane gene alone, irrespective of the sex of the animal. Initial pH (pH_{45}) values indicated highly significant differences ($P < 0.001$) between all the genotypes, with NN the highest (6.22), Nn intermediate (5.94), and nn the lowest (5.36). Drip loss values gave similar results ($P < 0.05$), with nn the highest (3.67%) and NN the lowest (1.53%), and Nn intermediate (2.30%). Drip loss values showed considerable differences ($P < 0.05$) between the halothane genotypes, with the nn genotypes having the highest value (3.67%) and NN the lowest value (1.53%). Objective colour evaluation (CIELAB) suggested that the presence of the gene is also accompanied by higher L* values, which indicate pale meat colour. Cooking losses, as well as Warner-Bratzler shear values, were higher with the inclusion of the halothane gene. The absence of genotype x sex interaction and the absence of sex differences indicate that the differences between these financially important characteristics are directly due to the presence of the halothane gene. Results from chemical analyses showed that the lean meat originating from the Nn genotypes had the highest moisture (73.0%) and lowest protein (21.0%) content, differing significantly ($P < 0.05$) from the NN (71.8% and 21.9%, respectively) and nn (71.8% and 22.3%, respectively) genotypes. Fat (NN = 2.02%, Nn = 1.84% and nn = 1.76%) and ash (NN = 1.38%, Nn = 1.33% and nn = 1.47%) values showed no significant differences between genotypes. The results are supported by other studies (Murray *et al.*, 1989; Jones *et al.*, 1988), showing that the presence of the halothane gene is accompanied by an increase in protein and lower intermuscular fat, with the Nn genotypes usually having intermediate values for fat and protein. These results not only provide further proof that nn genotypes deposit more lean meat than NN genotypes, but also that this advantage is at the cost of meat quality (as reflected by pH_{45} and L* values) and enhanced losses during chilling, deboning and cooking.

The strong correlation between pH_{45} and drip loss ($r = -0.77$), as well as between pH_{45} and reflectance (L*) values ($r = -0.67$) suggest that using these (pH_{45} and reflectance) variables in a regression model should give a good prediction of certain meat quality characteristics, such as drip loss. Regression coefficients calculated for drip loss using only pH_{45} and L* values resulted in $R^2=0.66$. The addition of more variables, such as pH_{24}, did not significantly improve R^2. This suggests that the implementation of these variables in a meat quality classification system would,

within limits, accurately predict moisture losses incurred during cutting and processing fresh meat, as well as assisting processors in selecting carcasses for processing or for fresh meat cuts.

12.3 Processing characteristics

Processing the meat derived from the different genotypes indicated that, even with the addition of certain water binding agents (rusk, phosphates), the presence of the halothane resulted in lower processing yields or inferior quality characteristics. Whole muscle products, such as back bacon, clearly showed the disadvantages associated with the halothane gene in the homozygous form, resulting in a significantly lower net gain (NN = 10.0%, Nn = 11.5% and nn = 3.4%). These losses occur in processing facilities and are rarely calculated, although it is conceivable that such losses are indirectly compensated for by increased prices to the consumer.

The results for canned hams indicated a similar pattern, with the inclusion of the halothane gene resulting in increased cooking losses for both hams without (NN = 27.92%, Nn = 30.12% and nn = 31.14%) and with phosphates (NN = 13.75%, Nn = 16.87% and nn = 17.73%). It is important to note that there were no significant differences ($P > 0.05$) between the Nn and nn genotypes for both treatments, emphasizing the contribution of the halothane gene to a reduction in water binding capacity. These losses are contained in the can, so there is no reduction in weight of the final product. However, product quality is compromised, since the hams derived from PSE meat would have a large percentage gelatinous cook out, and thus appear inferior. Although not evaluated, sensory characteristics could also suffer.

The results for the fresh sausage made from the meat originating from the three genotypes showed a similar pattern, in that inclusion of the halothane gene resulted in a higher total moisture loss. Sausage made without rusk showed significant differences ($P < 0.05$) in total moisture loss between the NN and nn genotypes (15.7% vs. 18.9%). The addition of rusk decreased the total moisture loss over all the genotypes, but still showed a similar pattern compared to the sausage with no rusk added (NN = 12.6%, Nn = 13.0% and nn = 14.2%). The sensory data on fresh sausages gave seemingly contradictory results, since those derived from the nn genotype were judged as being more juicy, which could be explained by the inability of the meat to retain moisture. These results are only contradictory if increased juiciness is considered a positive attribute, although too much juiciness may be interpreted by consumers as too much water added during manufacturing.

The emulsion products showed very little variation in cooking losses between genotypes (NN = 12.35%, Nn = 11.31% and nn = 12.47%), although some of the differences (Nn vs. nn) were statistically significant ($P < 0.05$). This suggest that the processes associated with manufacturing

emulsion products diminishes, to an extent, the poor meat quality associated with PSE meat, and that products processed to a higher degree are less susceptible to deviations due to poor meat quality.

Possible solutions to improve meat quality could include the use of variables, such as initial pH, to accurately predict meat quality in a production and processing environment. However, such a system must make it possible to identify environmental problem areas that could lead to poor meat quality, such as poor transport practices, holding facilities, preslaughter treatment and stunning practices. Pork producers must also be made fully aware of the impact of poor meat quality and its underlying factors, such as genotype and environmental conditions. This will ensure not only a better end product and higher financial returns, but could help in gaining a better market share for pork.

Annex A Table 1 Correlation values for meat quality characteristics

	WWT	CWT	pH_{45}	pH_{24}	L_f	a_f	b_f	chilloss	driploss	L_p	a_p	b_p	thaw	pump	bloss	bgain	fat	muscle
LWT	**0.87**	**0.86**	-0.19	-0.05	0.23	-0.11	0.09	-0.08	0.24	0.01	-0.03	-0.05	-0.03	-0.02	-0.01	-0.01	-0.03	**0.29**
WWT		**0.97**	-0.17	0.07	**0.30**	-0.02	0.19	-0.03	**0.26**	0.01	-0.07	-0.05	0.06	0.09	0.14	-0.02	-0.03	0.21
CWT			-0.16	0.02	**0.30**	< -0.01	0.20	**-0.26**	**0.28**	0.04	-0.08	0.01	0.10	0.11	0.11	< 0.01	-0.03	0.24
pH_{45}				**0.31**	**-0.67**	-0.21	< -0.01	-0.01	**-0.77**	**-0.27**	-0.20	-0.22	0.02	**0.32**	-0.04	**0.26**	**0.32**	**-0.59**
pH_{24}					**-0.28**	-0.20	-0.19	0.21	**-0.29**	**-0.48**	-0.21	**-0.43**	-0.09	**0.33**	0.01	0.23	0.15	**-0.39**
L_f						0.12	**0.54**	-0.06	**0.72**	0.22	0.12	0.31	0.01	-0.22	0.19	**-0.29**	-0.05	**0.42**
a_f							**0.52**	-0.09	**0.27**	0.09	**0.42**	0.24	0.19	0.08	0.10	-0.01	-0.21	0.08
b_f								-0.06	**0.41**	0.08	**0.28**	**0.28**	0.06	0.05	-0.01	0.05	0.07	0.17
chilloss									-0.11	< 0.01	-0.15	**0.31**	-0.18	-0.07	0.10	-0.12	< 0.01	-0.18
driploss										**0.31**	0.13	**0.35**	0.25	-0.08	-0.04	-0.03	-0.18	**0.47**
L_p											-0.16	**0.57**	0.09	-0.19	-0.05	-0.10	-0.09	**0.26**
a_p												**0.49**	< 0.01	-0.21	0.01	-0.16	-0.24	0.21
b_p													0.09	-0.16	-0.05	-0.08	-0.03	0.15
thaw														**0.72**	0.06	**0.48**	-0.02	0.01
pump															< 0.01	**0.72**	**0.28**	**-0.33**
bloss																**-0.69**	-0.24	0.11
bgain																	**0.36**	**-0.32**
fat																		**0.56**

LWT = live weight; WWT = warm carcass weight; CWT = cold carcass weight; pH_{45} = initial pH; pH_{24} = ultimate pH; Lf = L* of fresh sample; a_f = a* of fresh sample; b_f = b* of fresh sample; chilloss = % chilling loss; driploss = % drip loss; L_p = L* of bacon sample; a_p = a* of bacon sample; b_p = b* of bacon sample; thaw = % thawing loss of back (bacon); pump = % pumped yield of back (bacon); bloss = % moisture loss (bacon); bgain = % gain in back yield (bacon); fat = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; muscle = MLT thickness between 2-3 last thoracic vertebrae 45 mm from midline

Annex A Table 2 Correlation values for carcass quality characteristics

WWT	CWT	length	T1	T2-3	GM	fat	muscle	width	depth	area	LMP	total1	Fat1	bone1	lean
LWT **0.87**	**0.86**	**0.61**	0.08	0.08	-0.09	-0.03	**0.29**	0.16	**0.29**	**0.28**	**0.26**	**0.64**	0.19	**0.52**	**0.57**
WWT	**0.97**	**0.67**	0.09	0.08	-0.08	-0.03	0.21	**0.27**	0.20	**0.26**	**0.70**	**0.73**	0.22	**0.54**	**0.50**
CWT		**0.70**	0.14	0.07	-0.06	-0.03	0.24	**0.30**	0.24	**0.30**	**0.66**	**0.76**	**0.26**	**0.55**	**0.59**
length			-0.01	-0.22	-0.23	-0.24	**0.26**	**0.40**	**0.26**	**0.38**	**0.41**	**0.61**	0.16	**0.57**	**0.52**
T1				**0.54**	**0.62**	**0.61**	**-0.29**	**-0.50**	**-0.35**	**-0.44**	**-0.60**	0.20	**0.54**	-0.13	-0.08
T2-3					**0.71**	**0.72**	**-0.34**	**-0.50**	**-0.35**	**-0.55**	**-0.71**	0.07	**0.48**	-0.17	-0.14
GM						**0.72**	**-0.49**	**-0.59**	**-0.54**	**-0.55**	**-0.73**	-0.01	**0.60**	-0.23	**-0.31**
fat							**0.56**	**-0.65**	**-0.54**	**-0.53**	**-0.99**	-0.05	**0.55**	**-0.26**	**-0.34**
muscle								**0.52**	**0.91**	**0.78**	**0.66**	0.11	**-0.41**	0.22	**-0.36**
width									**0.51**	**0.75**	**0.67**	0.18	**-0.41**	**0.33**	**0.42**
depth										**0.83**	**0.63**	0.14	**-0.41**	0.25	**0.40**
area											**0.70**	0.15	**-0.53**	**0.30**	**0.47**
LMP												0.06	**-0.56**	**0.27**	**0.36**
total1													**0.47**	**0.71**	**0.82**
fat1														0.16	-0.06
bone1															**0.50**

LWT = live weight; WWT = warm carcass weight; CWT = cold carcass weight; length = carcass length; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. Gluteus medius*; fat = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; muscle = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; width = MLT width between 2-3 last thoracic vertebrae; depth = MLT depth between 2-3 last thoracic vertebrae; area = MLT area between 2-3 last thoracic vertebrae; LMP = percentage predicted lean yield; total1 = weight of shoulder; fat1 = weight of shoulder fat; bone1 = weight of bone in shoulder; lean = weight of lean in shoulder

Annex A Table 2 Correlation vales for carcass quality characteristics (continued)

	Total2	fat2	top	silver	thick	rump	ltrm	femur	pubis	llean	tibtr	thaw	pump	bloss	bgain
LWT	**0.53**	0.17	**0.36**	**0.38**	**0.38**	0.01	0.14	**0.29**	0.22	0.20	**0.40**	-0.03	-0.02	-0.01	-0.01
WWT	**0.54**	0.17	**0.49**	**0.48**	**0.44**	0.09	0.06	**0.36**	0.24	0.25	**0.36**	0.06	0.09	0.14	-0.02
CWT	**0.54**	0.17	**0.46**	**0.47**	**0.46**	0.03	0.10	**0.36**	0.23	**0.34**	**0.33**	0.10	0.11	0.11	<0.01
length	**0.51**	-0.06	**0.44**	**0.52**	**0.53**	0.05	0.10	**0.53**	**0.43**	**0.41**	**0.44**	0.14	-0.01	0.06	-0.05
T1	-0.20	**0.53**	**-0.37**	**-0.44**	-0.24	**-0.38**	-0.08	-0.18	-0.13	-0.12	**-0.31**	0.05	**0.29**	-0.11	**0.28**
T2-3	**0.29**	**0.69**	**-0.48**	**-0.57**	**-0.34**	-0.20	-0.13	**-0.33**	**-0.26**	**-0.32**	**-0.36**	-0.07	0.23	-0.12	0.25
GM	**-0.41**	**0.71**	**-0.56**	**-0.62**	**-0.44**	**-0.39**	**-0.30**	**-0.32**	**-0.27**	**-0.28**	**-0.33**	-0.04	0.23	-0.22	**0.32**
fat	**-0.51**	**0.70**	**-0.59**	**-0.68**	**-0.55**	**-0.47**	**-0.31**	-0.24	**-0.38**	**-0.34**	-0.24	-0.02	**0.28**	-0.24	**0.36**
muscle	**0.45**	**-0.44**	**0.40**	**0.55**	**0.45**	**0.48**	**0.58**	0.04	**0.33**	0.09	0.03	0.01	**-0.33**	0.11	**-0.32**
width	**0.53**	**-0.46**	**0.63**	**0.77**	**0.54**	**0.38**	0.17	**0.32**	**0.28**	**0.46**	**0.32**	0.06	**-0.27**	**0.29**	**-0.40**
depth	**0.46**	**-0.43**	**0.43**	**0.54**	**0.44**	**0.47**	**0.50**	0.08	0.24	0.12	0.02	0.10	**-0.28**	0.14	**-0.30**
area	**0.58**	**-0.54**	**0.56**	**0.72**	**0.57**	**0.43**	**0.49**	**0.27**	**0.27**	**0.31**	0.20	0.13	**-0.30**	0.01	-0.23
LMP	**0.53**	**-0.70**	**0.60**	**0.70**	**0.57**	**0.50**	**0.37**	0.23	**0.39**	**0.32**	0.22	0.02	**-0.30**	0.23	**-0.38**
total1	**0.55**	0.17	**0.40**	**0.31**	**0.49**	0.01	0.06	**0.33**	**0.26**	**0.30**	0.25	0.22	**0.34**	0.06	0.21
fat1	-0.16	**0.53**	**-0.29**	**-0.43**	-0.16	**-0.38**	-0.24	-0.04	< -0.01	-0.09	-0.15	0.02	**0.37**	0.03	0.25
bone1	**0.48**	-0.15	**0.47**	**0.51**	**0.44**	-0.02	-0.13	**0.45**	**0.30**	**0.30**	**0.51**	0.11	0.11	0.11	0.01
lean	**0.70**	-0.12	**0.56**	**0.54**	**0.52**	**0.28**	**0.29**	**0.29**	0.24	**0.36**	0.23	**0.26**	0.18	0.06	0.09
total2		-0.13	**0.75**	**0.83**	**0.81**	**0.53**	0.24	**0.36**	**0.49**	**0.48**	**0.37**	0.02	-0.23	0.21	**-0.31**
fat2			**-0.46**	**-0.51**	**-0.32**	-0.19	-0.24	-0.17	-0.16	**-0.29**	-0.25	-0.15	0.15	-0.15	0.21
top				**0.80**	**0.56**	**0.33**	0.04	0.29	**0.27**	**0.44**	**0.49**	0.13	-0.10	**0.34**	**-0.34**
slver					**0.71**	**0.39**	0.15	**0.32**	**0.42**	**0.49**	**0.46**	0.04	**-0.28**	0.24	**-0.37**
thick						**0.49**	**0.37**	**0.29**	**0.56**	**0.38**	0.20	0.06	-0.25	0.12	-0.27
rump							**0.46**	**0.54**	**0.29**	-0.16	-0.21	-0.01	**-0.29**	0.17	**-0.33**
ltrm								0.12	**0.26**	-0.13	**-0.34**	-0.01	-0.26	-0.15	-0.09
femur									**0.48**	0.19	**0.46**	**0.29**	0.16	0.06	0.08
pubis										0.08	0.18	0.14	-0.08	0.19	-0.18
llean											**0.46**	0.10	-0.01	0.08	-0.06
tibtr												-0.03	-0.06	0.13	-0.13
thaw													**0.72**	0.06	**0.48**
pump														< 0.01	**0.73**
bloss															**-0.69**

LWT = live weight; WWT = warm carcass weight; CWT = cold carcass weight; length = carcass length; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*; fat = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; muscle = muscle thickness between 2-3 last thoracic vertebrae 45 mm from midline; width = MLT width between 2-3 last thoracic vertebrae; depth = MLT depth between 2-3 last thoracic vertebrae; area = MLT area between 2-3 last thoracic vertebrae 45 mm from midline; LMP = percentage predicted lean yield; total1 = weight of shoulder; fat1 = weight of shoulder fat; bone1 = weight of bone in shoulder; lean = weight of lean in shoulder; total2 = weight of leg; fat2 = weight of fat in leg; top = weight of topside; slver = weight of silverside; thick = weight of thickflank; rump = weight of rump; ltrm = weight of lean trim on leg; femur = weight of femur; pubis = weight of pubis; llean = weight of lean on lower leg; tibtars = weight of tibiotarsus; thaw = % thawing loss of back (bacon); pump = % pumped yield of back (bacon); bloss = % moisture loss (bacon); bgain = % gain in back yield (bacon)

Annex B Table 1. Regression Models for prediction of Dependent Variable: Drip loss

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	PH45	PH24	FAT	MSCLE	L1	A1	B1
1	0.59418	11.181	0.8911	16.9603	.	.	-2.4775	.	.	.	.	.	.
1	0.51248	24.706	0.9767	-13.1446	.	.	.	.	.	.	0.3562	.	.
1	0.22425	72.419	1.2320	-3.9094	.	.	.	.	.	0.1076	.	.	.
1	0.16836	81.672	1.2756	-1.4321	.	.	.	.	.	.	.	.	0.5484
1	0.08788	95.0	1.3359	15.5083	.	.	.	-2.3663	.	.	.	.	.
1	0.07625	96.9	1.3444	-4.4350	.	0.1040	.	.	.	.	.	.	.
1	0.07210	97.6	1.3474	0.0809	.	.	.	.	.	.	.	0.4048	.
1	0.06701	98.4	1.3511	-4.2122	0.0983	.	.	.	.	.	.	.	.
1	0.03412	103.9	1.3747	3.3201	.	.	.	.	-0.0611	.	.	.	.
2	0.66456	1.529	0.8172	4.5860	.	.	-1.6981	.	.	.	0.1789	.	.
2	0.61797	9.242	0.8721	12.6483	.	0.0589	-2.3968	.	.	.	.	.	.
2	0.61294	10.074	0.8778	14.5806	.	.	-2.3038	.	.	.	.	.	0.1968
2	0.61111	10.377	0.8799	13.1924	0.0502	.	-2.4056	.	.	.	.	.	.
2	0.60541	11.322	0.8863	15.6062	.	.	-2.4029	.	.	.	.	0.1635	.
3	0.67788	1.324	0.8079	2.9201	.	.	-1.6048	.	.	.	0.1816	0.1782	.
3	0.67221	2.263	0.8150	3.1660	.	0.0346	-1.7209	.	.	.	0.1628	.	.
3	0.66896	2.802	0.8191	3.3981	0.0264	.	-1.7103	.	.	.	0.1674	.	.
3	0.66578	3.328	0.8230	6.1659	.	.	-1.6717	-0.2939	.	.	0.1765	.	.
3	0.66527	3.412	0.8236	4.6591	.	.	-1.6976	.	.	.	0.1704	.	0.0422
4	0.68611	1.962	0.8048	1.4100	.	0.0359	-1.6263	.	.	.	0.1650	0.1821	.
4	0.68319	2.445	0.8085	1.5531	0.0290	.	-1.6149	.	.	.	0.1691	0.1845	.
4	0.68285	2.502	0.8089	1.7169	.	.	-1.5540	.	.	.	0.2136	0.2787	-0.1523
4	0.67823	3.267	0.8148	2.3789	.	.	-1.5625	.	.	0.00524	0.1811	0.1799	.
4	0.67822	3.268	0.8148	3.7979	.	.	-1.5929	-0.1561	.	.	0.1803	0.1741	.
5	0.69225	2.946	0.8042	-0.0430	.	0.0385	-1.5712	.	.	.	0.1994	0.2946	-0.1700
5	0.69008	3.304	0.8070	2.0916	-0.1044	0.1365	-1.6505	.	.	.	0.1633	0.1704	.
5	0.68928	3.438	0.8081	0.0699	0.0321	.	-1.5594	.	.	.	0.2033	0.2970	-0.1694
5	0.68686	3.839	0.8112	2.6815	.	0.0369	-1.6091	-0.2340	.	.	0.1625	0.1761	.
5	0.68658	3.884	0.8116	1.6169	.	0.0363	-1.6643	.	0.00788	.	0.1612	0.1871	.
6	0.69558	4.394	0.8074	0.6575	-0.0958	0.1307	-1.5962	.	.	.	0.1962	0.2780	-0.1612
6	0.69458	4.559	0.8087	0.1503	.	0.0400	-1.6491	.	0.0185	.	0.1975	0.3287	-0.2041
6	0.69274	4.864	0.8111	1.0227	.	0.0393	-1.5582	-0.1912	.	.	0.1968	0.2875	-0.1668
6	0.69227	4.942	0.8117	0.0773	.	0.0389	-1.5823	.	.	-0.00138	0.1996	0.2948	-0.1709
6	0.69152	5.067	0.8127	0.2464	0.0337	.	-1.6354	.	0.0181	.	0.2015	0.3307	-0.2031

7	0.69777	6.032	0.8121	0.8294	-0.0937	0.1302	-1.6711	.	0.0179	.	0.1944	0.3114	-0.1945
7	0.69584	6.351	0.8147	1.1087	-0.1011	0.1370	-1.6356	.	.	-0.00475	0.1965	0.2779	-0.1639
7	0.69565	6.383	0.8150	1.0482	-0.0923	0.1277	-1.5902	-0.0747	.	.	0.1953	0.2759	-0.1603
7	0.69520	6.458	0.8156	1.3420	.	0.0410	-1.6367	-0.2129	0.0190	.	0.1946	0.3217	-0.2015
7	0.69518	6.461	0.8156	-0.5078	.	0.0383	-1.6074	.	0.0243	0.00827	0.1960	0.3380	-0.2093
8	0.69789	8.011	0.8199	1.3530	-0.0890	0.1261	-1.6642	-0.0997	0.0182	.	0.1932	0.3090	-0.1937
8	0.69789	8.012	0.8199	0.4900	-0.0892	0.1250	-1.6506	.	0.0206	0.00385	0.1938	0.3166	-0.1973
8	0.69601	8.323	0.8224	1.8376	-0.0966	0.1335	-1.6346	-0.1204	.	-0.00579	0.1951	0.2744	-0.1631
8	0.69550	8.408	0.8231	0.5658	.	0.0395	-1.6081	-0.1628	0.0232	0.00623	0.1941	0.3304	-0.2060
8	0.69292	8.835	0.8266	0.4606	0.0337	.	-1.5819	-0.1718	0.0247	0.00877	0.1971	0.3356	-0.2070
9	0.69796	10.000	0.8280	0.9793	-0.0864	0.1229	-1.6497	-0.0789	0.0202	0.00300	0.1930	0.3136	-0.1961

WWT = warm carcass weight; CWT = cold carcass weight; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline L1 = L* of fresh sample; A1 = a* of fresh sample; B1 = b* of fresh sample

Annex B Table 2. Regression Models for prediction of Dependent Variable: Final bacon yield

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	CHLOSS	PH45	PH24	FAT	MSCLE	L1	A1	B1
1	0.13254	9.943	7.5338	-2.5161	.	.	.	.	.	0.6965	.	.	.	.
1	0.10302	12.187	7.6609	33.1316	.	.	.	.	.	.	-0.4218	.	.	.
1	0.08699	13.406	7.7291	45.5250	.	.	.	.	.	.	.	-0.8487	.	.
1	0.06784	14.862	7.8097	-19.9351	.	.	.	4.8409	.	.	.	.	.	.
1	0.05418	15.900	7.8667	-51.1569	.	.	.	.	10.7448	.	.	.	.	.
1	0.01543	18.846	8.0263	10.4049	.	.	-0.7910	.	.	.	.	.	.	.
1	0.00212	19.858	8.0803	6.1792	.	.	.	.	.	.	.	.	.	0.3557
1	0.00060	19.973	8.0865	12.2075	-0.0537	.	.	.	.	.	.	.	.	.
1	0.00014	20.008	8.0883	9.2006	.	.	.	.	.	.	.	.	-0.1026	.
1	0.00000	20.019	8.0889	8.3937	.	0.00358	.	.	.	.	.	.	.	.
2	0.20987	6.064	7.2529	32.7138	.	.	.	.	.	0.6714	.	-0.8011	.	.
2	0.16509	9.469	7.4556	-48.5434	.	.	.	.	8.4223	0.6443	.	.	.	.
2	0.15580	10.175	7.4970	-18.5896	.	.	.	2.9899	.	0.5985	.	.	.	.
2	0.15245	10.430	7.5119	13.4172	.	.	.	.	.	0.5137	-0.2239	.	.	.
2	0.14798	10.770	7.5317	-0.7384	.	.	-0.7911	.	.	0.6965	.	.	.	.
3	0.25224	4.844	7.1185	37.0961	.	.	.	.	.	0.6268	.	-1.1867	.	1.9021
3	0.23003	6.532	7.2235	35.8808	.	.	-0.9059	.	.	0.6706	.	-0.8269	.	.
3	0.22160	7.173	7.2629	0.0764	.	.	.	.	5.2560	0.6416	.	-0.7121	.	.
3	0.22004	7.292	7.2702	21.6187	.	0.2302	.	.	.	0.6748	.	-0.8923	.	.
3	0.21908	7.365	7.2747	28.6581	.	.	.	.	.	0.7091	.	-0.8322	0.8599	.
4	0.27036	5.466	7.0954	39.9979	.	.	-0.8596	.	.	0.6271	.	-1.2020	.	1.8571
4	0.26697	5.724	7.1119	0.6001	.	.	.	.	5.9035	0.5918	.	-1.1009	.	1.9723
4	0.26064	6.205	7.1425	26.9066	.	0.2096	.	.	.	0.6308	.	-1.2619	.	1.8641
4	0.25728	6.460	7.1587	42.8783	.	.	.	.	.	0.5717	.	-1.2879	-0.9032	2.5626
4	0.25648	6.521	7.1625	29.5227	0.1499	.	.	.	.	0.6299	.	-1.2400	.	1.8801
5	0.29356	5.703	7.0460	-6.1096	.	.	-1.0639	.	7.5698	0.5822	.	-1.0957	.	1.9365
5	0.28981	5.988	7.0647	87.0838	33.2753	-34.0387	-23.8986	.	.	0.7077	.	-0.8310	.	.
5	0.27729	6.940	7.1267	46.9571	.	.	-0.9066	.	.	0.5623	.	-1.3219	-1.0623	2.6316
5	0.27435	7.163	7.1412	32.6405	0.1452	.	-0.8535	.	.	0.6301	.	-1.2535	.	1.8362
5	0.27386	7.200	7.1436	32.8612	.	0.1398	-0.7594	.	.	0.6297	.	-1.2504	.	1.8371
5	0.27357	7.222	7.1450	55.3839	.	.	-0.8860	-1.5551	.	0.6737	.	-1.3560	.	1.8282
6	0.33431	4.605	6.9040	95.1	34.8156	-35.6399	-24.9326	.	.	0.6633	.	-1.2163	.	1.9545
6	0.30419	6.895	7.0585	84.6402	35.4813	-36.2863	-25.3484	.	.	0.7581	.	-0.8721	1.0904	.
6	0.30347	6.949	7.0621	51.6959	32.2588	-33.0435	-23.3892	.	5.9030	0.6723	.	-0.7187	.	.
6	0.29986	7.224	7.0804	12.1757	.	.	-1.1167	-2.2006	8.1422	0.6448	.	-1.3057	.	1.9016
6	0.29761	7.395	7.0918	1.8291	.	.	-1.0888	.	7.1481	0.5347	.	-1.1941	-0.8198	2.5298

7	0.35119	5.322	6.8811	55.9681	33.7411	-34.5915	-24.4041	.	6.5752	0.6222	.	-1.1057	.	2.0279
7	0.33699	6.401	6.9560	98.3	35.1099	-35.9293	-25.2010	.	.	0.5878	-0.0966	-1.1363	.	1.9421
7	0.33624	6.458	6.9600	98.1	33.9872	-34.8027	-24.3929	.	.	0.6274	.	-1.2767	-0.5714	2.3703
7	0.33465	6.579	6.9683	99.2	34.3621	-35.1713	-24.6246	-0.5151	.	0.6783	.	-1.2687	.	1.9431
7	0.32154	7.576	7.0366	44.1686	34.6118	-35.4464	-24.9570	.	6.6986	0.7245	.	-0.7500	1.2307	.
8	0.35265	7.211	6.9405	62.9216	32.7473	-33.5673	-23.7380	-1.0752	6.8639	0.6518	.	-1.2102	.	2.0071
8	0.35213	7.251	6.9433	59.0194	33.1886	-34.0322	-24.0398	.	6.4128	0.5981	.	-1.1507	-0.3994	2.3167
8	0.35145	7.302	6.9469	58.4302	33.8747	-34.7226	-24.5097	.	6.3383	0.5993	-0.0312	-1.0839	.	2.0212
8	0.33972	8.194	7.0094	102.4	34.1592	-34.9672	-24.5931	.	.	0.5332	-0.1112	-1.1967	-0.6865	2.4398
8	0.33844	8.291	7.0162	108.3	34.1958	-34.9808	-24.5980	-1.1255	.	0.6010	-0.1218	-1.2298	.	1.9138
9	0.35391	9.115	7.0027	67.3175	31.9824	-32.7908	-23.2325	-1.2012	6.7071	0.6270	.	-1.2752	-0.4687	2.3437
9	0.35350	9.146	7.0049	69.3795	32.7556	-33.5631	-23.7743	-1.3435	6.4816	0.6153	-0.0598	-1.1943	.	1.9892
9	0.35262	9.213	7.0097	62.8932	33.3004	-34.1395	-24.1381	.	6.0580	0.5627	-0.0437	-1.1262	-0.4542	2.3470
9	0.34218	10.007	7.0660	116.4	32.7572	-33.5172	-23.6721	-1.4979	.	0.5401	-0.1476	-1.3330	-0.8204	2.4992
9	0.32294	11.469	7.1686	54.6164	33.6007	-34.3987	-24.2976	-1.1230	6.7049	0.7267	-0.0339	-0.8390	1.1577	.
10	0.35542	11.000	7.0655	77.3233	31.7890	-32.5773	-23.1468	-1.6018	6.1425	0.5705	-0.0817	-1.2709	-0.5942	2.4092

WWT = warm carcass weight; CWT = cold carcass weight; CHLOSS = % chilling loss; pH45= initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; L1 = L* of fresh sample; A1 = a* of fresh sample; B1 = b* of fresh sample

Annex B Table 3. Regression Models for prediction of Dependent Variable: Leg fat as a percentage of cold carcass weight

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	LNGTH	PH45	PH24	FAT	MSCLE	T1	T2-3	GM
1	0.52668	14.080	0.52582	2.8424	.	.	.	.	.	.	.	.	.	0.1157
1	0.49342	19.004	0.54398	2.8959	.	.	.	.	.	0.1270	.	.	.	.
1	0.43744	27.293	0.57325	2.6360	.	.	.	.	.	.	.	.	0.1202	.
1	0.31271	45.760	0.63362	2.3085	.	.	.	.	.	.	.	0.0854	.	.
1	0.26911	52.216	0.65341	8.6694	.	.	.	.	.	.	-0.0644	.	.	.
1	0.16031	68.325	0.70036	0.7786	.	.	.	0.7032	.	.	.	.	.	.
1	0.09918	77.376	0.72541	13.4323	.	.	-0.0110	.	.	.	.	.	.	.
1	0.07700	80.659	0.73428	-1.8066	.	.	.	.	1.2103	.	.	.	.	.
1	0.03625	86.693	0.75031	7.4806	.	-0.0392	.	.	.	.	.	.	.	.
1	0.03178	87.354	0.75205	7.3940	-0.0370	.	.	.	.	.	.	.	.	.
2	0.59237	6.353	0.49223	2.5410	.	.	.	.	.	0.0672	.	.	.	0.0727
2	0.57071	9.560	0.50514	2.4171	.	.	.	.	.	.	.	.	0.0539	0.0823
2	0.56633	10.209	0.50771	0.8598	.	.	.	0.3653	.	.	.	.	.	0.1061
2	0.56089	11.015	0.51089	4.6771	.	.	.	.	.	.	-0.0264	.	.	0.0990
2	0.55056	12.544	0.51686	7.2305	.	.	-0.00556	.	.	.	.	.	.	0.1099
3	0.61765	4.610	0.48096	0.9672	.	.	.	0.2957	.	0.0602	.	.	.	0.0694
3	0.61473	5.043	0.48279	4.5749	.	-0.0308	.	.	.	0.0681	.	.	.	0.0706
3	0.60959	5.805	0.48601	4.3867	-0.0273	.	.	.	.	0.0683	.	.	.	0.0704
3	0.60864	5.945	0.48660	6.2003	.	.	-0.00462	.	.	0.0636	.	.	.	0.0703
3	0.60656	6.253	0.48789	-0.3539	.	.	.	.	0.5343	0.0681	.	.	.	0.0676
4	0.63365	4.242	0.47505	2.9043	.	-0.0264	.	0.2589	.	0.0619	.	.	.	0.0680
4	0.63340	4.278	0.47521	4.6532	.	-0.0356	.	.	.	0.0509	.	.	0.0394	0.0569
4	0.63015	4.760	0.47731	1.6114	.	-0.0317	.	.	0.5574	0.0691	.	.	.	0.0652
4	0.62942	4.868	0.47779	4.2395	.	.	-0.00396	0.2702	.	0.0577	.	.	.	0.0676
4	0.62907	4.919	0.47801	2.6642	-0.0226	.	.	0.2631	.	0.0619	.	.	.	0.0679
5	0.64906	3.961	0.46924	0.7833	.	-0.0391	.	.	0.7057	0.0572	.	0.0297	.	0.0520
5	0.64584	4.437	0.47139	1.9729	.	-0.0360	.	.	0.5030	0.0531	.	.	0.0363	0.0531
5	0.64499	4.563	0.47195	5.7406	.	-0.0361	.	.	.	.	-0.0214	0.0283	0.0549	0.0481
5	0.64377	4.743	0.47276	3.3410	.	-0.0310	.	0.2006	.	0.0500	.	.	0.0304	0.0580
5	0.64371	4.753	0.47280	4.6235	.	-0.0406	.	.	.	0.0425	.	0.0213	0.0388	0.0487
6	0.66319	3.868	0.46401	1.1607	.	-0.0428	.	.	0.6479	0.0426	.	0.0285	0.0345	0.0411
6	0.65696	4.791	0.46829	0.8915	-0.0395	.	.	.	0.6779	0.0441	.	0.0253	0.0351	0.0414
6	0.65547	5.012	0.46930	0.4163	.	-0.0352	.	0.1605	0.5809	0.0547	.	0.0260	.	0.0531
6	0.65372	5.270	0.47049	3.0088	.	-0.0397	.	.	0.4650	.	-0.0160	0.0337	0.0520	0.0456
6	0.65328	5.335	0.47079	5.3873	.	-0.0359	.	.	.	0.0299	-0.0157	0.0229	0.0433	0.0432

7	0.66569	5.498	0.46671	1.2012	0.0481	-0.0895	.	.	0.6028	0.0416	.	0.0311	0.0328	0.0420
7	0.66560	5.511	0.46677	0.8816	.	-0.0399	.	0.1025	0.5751	0.0427	.	0.0262	0.0304	0.0431
7	0.66538	5.543	0.46692	2.0540	.	-0.0400	.	.	0.5552	0.0360	-0.00815	0.0283	0.0375	0.0393
7	0.66332	5.850	0.46837	1.4631	.	-0.0404	-0.0006	.	0.6498	0.0423	.	0.0287	0.0337	0.0411
7	0.66052	6.263	0.47031	0.4729	0.0681	-0.1013	.	0.1728	0.5031	0.0520	.	0.0293	.	0.0537
8	0.66880	7.037	0.46906	0.8873	0.0548	-0.0927	.	0.1173	0.5130	0.0416	.	0.0289	0.0278	0.0444
8	0.66740	7.245	0.47006	1.9912	0.0434	-0.0825	.	.	0.5247	0.0358	-0.00724	0.0307	0.0356	0.0404
8	0.66617	7.427	0.47092	1.5139	.	-0.0391	.	0.0699	0.5418	0.0386	-0.00496	0.0268	0.0335	0.0414
8	0.66578	7.485	0.47120	1.4526	0.0477	-0.0871	-0.0005	.	0.6046	0.0414	.	0.0312	0.0321	0.0420
8	0.66577	7.487	0.47121	1.2234	.	-0.0371	-0.00068	0.1033	0.5766	0.0424	.	0.0265	0.0295	0.0431
9	0.66894	9.017	0.47364	1.2028	0.0523	-0.0899	.	0.1004	0.4993	0.0396	-0.00248	0.0291	0.0295	0.0435
9	0.66892	9.020	0.47365	1.1774	0.0544	-0.0900	-0.00058	0.1180	0.5147	0.0413	.	0.0290	0.0270	0.0445
9	0.66749	9.231	0.47467	2.2534	0.0431	-0.0800	-0.00051	.	0.5264	0.0356	-0.00726	0.0308	0.0349	0.0404
9	0.66633	9.404	0.47550	1.8405	.	-0.0364	-0.00066	0.0710	0.5435	0.0384	-0.00492	0.0270	0.0326	0.0414
9	0.66218	10.018	0.47845	0.6885	0.0696	-0.0981	-0.00152	0.1974	0.5368	0.0532	0.00494	0.0293	.	0.0538
10	0.66905	11.000	0.47836	1.4879	0.0519	-0.0872	-0.00057	0.1011	0.5010	0.0394	-0.00246	0.0292	0.0287	0.0436

WWT = warm carcass weight; CWT = cold carcass weight; LNGTH = carcass length; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*

Annex B Table 4. Regression Models for prediction of Dependent Variable: Lean in leg as a percentage of cold carcass weight

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	LNGTH	PH45	PH24	FAT	MSCLE	T1	T2-3	GM
1	0.61295	32.873	0.9244	18.8003	.	.	.	.	.	-0.2751	.	.	.	.
1	0.47167	65.313	1.0800	18.2343	.	.	.	.	.	.	.	.	.	-0.2128
1	0.38421	85.395	1.1659	18.5732	.	.	.	.	.	.	.	.	-0.2189	.
1	0.32198	99.7	1.2234	19.5648	.	.	.	.	.	.	.	-0.1686	.	.
1	0.31507	101.3	1.2297	6.5272	.	.	.	.	.	.	0.1355	.	.	.
1	0.19206	129.5	1.3355	35.0737	.	.	.	.	-3.7159	.	.	.	.	.
1	0.18074	132.1	1.3448	22.9624	.	.	.	-1.4514	.	.	.	.	.	.
1	0.02328	168.3	1.4684	6.3881	.	.	0.0104	.	.	.	.	.	.	.
1	0.00244	173.1	1.4840	15.6859	.	-0.0198	.	.	.	.	.	.	.	.
1	0.00031	173.5	1.4856	14.8753	-0.00715	.	.	.	.	.	.	.	.	.
2	0.71854	10.629	0.7951	34.0277	.	.	.	.	-2.7864	-0.2579	.	.	.	.
2	0.64739	26.966	0.8900	22.3931	.	.	.	-0.6683	.	-0.2532	.	.	.	.
2	0.64305	27.962	0.8954	19.1808	.	.	.	.	.	-0.2110	.	.	.	-0.0780
2	0.63476	29.866	0.9058	15.7369	.	.	.	.	.	-0.2400	0.0431	.	.	.
2	0.62510	32.084	0.9177	19.5895	.	.	.	.	.	-0.2450	.	-0.0415	.	.
3	0.74396	6.791	0.7651	36.2871	.	.	.	.	-2.9886	-0.2126	.	-0.0606	.	.
3	0.73202	9.533	0.7828	33.2764	.	.	.	.	-2.6015	-0.2154	.	.	.	-0.0531
3	0.72827	10.393	0.7882	34.6715	.	.	.	-0.3701	-2.5401	-0.2473	.	.	.	.
3	0.72298	11.609	0.7959	35.7103	.	-0.0267	.	.	-2.7740	-0.2587	.	.	.	.
3	0.71962	12.379	0.8007	35.8503	.	.	-0.00231	.	-2.7857	-0.2607	.	.	.	.
4	0.76383	4.229	0.7415	36.5763	0.3624	-0.3758	.	.	-3.1931	-0.2551	.	.	.	.
4	0.74940	7.543	0.7638	36.6022	.	.	.	-0.2802	-2.7867	-0.2080	.	-0.0560	.	.
4	0.74805	7.852	0.7659	35.5030	.	.	.	.	-2.8492	-0.1944	.	-0.0515	.	-0.0312
4	0.74513	8.523	0.7703	34.9004	.	.	.	.	-2.8730	-0.2043	0.0107	-0.0610	.	.
4	0.74510	8.529	0.7703	37.0555	.	-0.0138	.	.	-2.9729	-0.2151	.	-0.0579	.	.
5	0.77396	3.902	0.7321	37.4045	0.3150	-0.3211	.	.	-3.2775	-0.2250	.	-0.0405	.	.
5	0.77325	4.066	0.7332	36.0572	0.3412	-0.3580	.	.	-3.0112	-0.2195	.	.	.	-0.0448
5	0.77120	4.537	0.7366	37.4830	0.3419	-0.3621	.	-0.3298	-2.9471	-0.2460	.	.	.	.
5	0.76826	5.211	0.7413	34.2562	0.3711	-0.3926	.	.	-2.9592	-0.2388	0.0220	.	.	.
5	0.76506	5.947	0.7464	34.7222	0.3670	-0.3944	0.00350	.	-3.1929	-0.2512	.	.	.	.
6	0.78039	4.426	0.7284	36.9561	0.3212	-0.3446	.	-0.3247	-2.7712	-0.2110	.	.	.	-0.0443
6	0.77870	4.815	0.7312	38.0394	0.3042	-0.3168	.	-0.2687	-3.0664	-0.2214	.	-0.0354	.	.
6	0.77821	4.926	0.7320	36.8239	0.3115	-0.3219	.	.	-3.1255	-0.2069	.	-0.0304	.	-0.0323
6	0.77769	5.046	0.7328	35.2462	0.3245	-0.3384	.	.	-3.0599	-0.2111	0.0202	-0.0391	.	.
6	0.77667	5.280	0.7345	34.0383	0.3499	-0.3737	.	.	-2.8144	-0.2070	0.0194	.	.	-0.0425

7	0.78348	5.717	0.7302	37.4583	0.2999	-0.3174	.	-0.2838	-2.8931	-0.2020	.	-0.0244	.	-0.0343
7	0.78174	6.116	0.7331	37.2751	0.3110	-0.3383	.	-0.3687	-2.7619	-0.2193	.	.	0.0218	-0.0517
7	0.78142	6.190	0.7336	34.8528	0.3206	-0.3380	.	.	-2.9325	-0.1951	0.0188	-0.0297	.	-0.0303
7	0.78109	6.265	0.7342	35.5525	0.3252	-0.3592	0.00265	-0.3214	-2.7765	-0.2087	.	.	.	-0.0435
7	0.78087	6.316	0.7346	35.9826	0.3276	-0.3531	.	-0.2808	-2.7206	-0.2069	0.00818	.	.	-0.0434
8	0.78491	7.389	0.7349	37.7929	0.2892	-0.3105	.	-0.3286	-2.8851	-0.2105	.	-0.0247	0.0225	-0.0418
8	0.78437	7.511	0.7358	35.8843	0.3038	-0.3329	0.00300	-0.2787	-2.9032	-0.1991	.	-0.0252	.	-0.0331
8	0.78414	7.565	0.7362	36.3254	0.3068	-0.3265	.	-0.2307	-2.8373	-0.1968	0.0097	-0.0252	.	-0.0329
8	0.78303	7.819	0.7380	35.3906	0.3143	-0.3570	0.00370	-0.3744	-2.7672	-0.2181	.	.	0.0269	-0.0524
8	0.78276	7.883	0.7385	32.8645	0.3255	-0.3575	0.00367	.	-2.9321	-0.1910	0.0195	-0.0306	.	-0.0288
9	0.78649	9.024	0.7394	35.7242	0.2918	-0.3300	0.00411	-0.3330	-2.8969	-0.2087	.	-0.0259	0.0281	-0.0420
9	0.78519	9.323	0.7417	34.4809	0.3118	-0.3445	0.00327	-0.2190	-2.8418	-0.1931	0.0108	-0.0261	.	-0.0314
9	0.78502	9.363	0.7420	37.2301	0.2938	-0.3156	.	-0.2983	-2.8607	-0.2070	0.00442	-0.0250	0.0195	-0.0401
9	0.78308	9.809	0.7453	35.0543	0.3172	-0.3602	0.00369	-0.3566	-2.7516	-0.2161	0.00264	.	0.0251	-0.0515
9	0.78288	9.855	0.7457	32.8849	0.3228	-0.3563	0.00393	.	-2.9495	-0.1940	0.0187	-0.0310	0.00661	-0.0309
10	0.78660	11.000	0.7467	35.1837	0.2962	-0.3348	0.00410	-0.3037	-2.8732	-0.2053	0.00428	-0.0262	0.0252	-0.0405

WWT = warm carcass weight; CWT = cold carcass weight; LNGTH = carcass length; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*

Annex B Table 5. Regression Models for prediction of Dependent Variable: Topside as a percentage of cold carcass weight

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	LNGTH	PH45	PH24	FAT	MSCLE	T1	T2-3	GM
1	0.40123	6.933	0.40840	5.7649	.	.	.	.	.	-0.0791	.	.	.	.
1	0.34356	12.995	0.42761	5.6627	.	.	.	.	.	.	.	.	.	-0.0645
1	0.32278	15.178	0.43433	5.8591	.	.	.	.	.	.	.	.	-0.0713	.
1	0.22532	25.422	0.46453	6.0350	.	.	.	.	.	.	.	-0.0501	.	.
1	0.11024	37.517	0.49784	2.8460	.	.	.	.	.	.	0.0285	.	.	.
1	0.08507	40.163	0.50483	9.3875	.	.	.	.	-0.8784	.	.	.	.	.
1	0.08383	40.293	0.50517	6.5716	.	.	.	-0.3511	.	.	.	.	.	.
1	0.02043	46.957	0.52236	1.8345	.	.	0.00346	.	.	.	.	.	.	.
1	0.00382	48.702	0.52677	3.9095	0.00886	.	.	.	.	.	.	.	.	.
1	0.00032	49.071	0.52769	4.3351	.	0.00253	.	.	.	.	.	.	.	.
2	0.44094	4.760	0.39807	9.0820	.	.	.	.	-0.6070	-0.0753	.	.	.	.
2	0.43530	5.352	0.40007	5.9087	.	.	.	.	.	-0.0548	.	.	.	-0.0295
2	0.42679	6.247	0.40308	5.9844	.	.	.	.	.	-0.0582	.	.	-0.0290	.
2	0.41309	7.687	0.40786	6.0419	.	.	.	.	.	-0.0685	.	-0.0146	.	.
2	0.40983	8.030	0.40899	6.4026	.	.	.	-0.1186	.	-0.0752	.	.	.	.
3	0.46366	4.371	0.39336	8.7356	.	.	.	.	-0.5217	-0.0557	.	.	.	-0.0245
3	0.46041	4.713	0.39455	9.7844	.	.	.	.	-0.6698	-0.0612	.	-0.0189	.	.
3	0.45768	5.000	0.39555	8.9097	.	.	.	.	-0.5425	-0.0586	.	.	-0.0238	.
3	0.45298	5.494	0.39726	10.6826	.	.	.	.	-0.7405	-0.0845	-0.0122	.	.	.
3	0.44456	6.379	0.40030	6.0206	.	.	.	.	.	-0.0465	.	.	-0.0189	-0.0231
4	0.49719	2.847	0.38431	9.4206	0.1511	-0.1452	.	.	-0.7818	-0.0738	.	.	.	.
4	0.47909	4.749	0.39117	10.5284	.	.	.	.	-0.6670	-0.0646	-0.0139	.	.	-0.0264
4	0.47218	5.476	0.39376	9.3120	.	.	.	.	-0.5858	-0.0503	.	-0.0133	.	-0.0188
4	0.47155	5.542	0.39399	11.3079	.	.	.	.	-0.7968	-0.0704	-0.0118	-0.0184	.	.
4	0.47103	5.597	0.39418	9.5359	.	.	.	.	-0.6077	-0.0498	.	-0.0159	-0.0194	.
5	0.51771	2.691	0.37986	9.0749	0.1558	-0.1471	.	.	-0.7164	-0.0550	.	.	-0.0266	.
5	0.51233	3.255	0.38197	9.1868	0.1415	-0.1371	.	.	-0.6999	-0.0578	.	.	.	-0.0202
5	0.50700	3.816	0.38405	10.6467	0.1465	-0.1363	.	.	-0.9054	-0.0824	-0.0116	.	.	.
5	0.50456	4.073	0.38500	9.6714	0.1367	-0.1286	.	.	-0.8074	-0.0647	.	-0.0123	.	.
5	0.49724	4.842	0.38783	9.4484	0.1504	-0.1447	.	-0.0101	-0.7743	-0.0735	.	.	.	.
6	0.52444	3.984	0.38074	10.5352	0.1357	-0.1266	.	.	-0.8314	-0.0662	-0.0130	.	.	-0.0217
6	0.52342	4.090	0.38115	10.0561	0.1518	-0.1401	.	.	-0.8182	-0.0634	-0.00900	.	-0.0241	.
6	0.52307	4.128	0.38129	8.9985	0.1486	-0.1415	.	.	-0.6777	-0.0487	.	.	-0.0209	-0.0130
6	0.52107	4.338	0.38209	9.2769	0.1455	-0.1355	.	.	-0.7395	-0.0503	.	-0.00846	-0.0244	.
6	0.51887	4.569	0.38296	8.9180	0.1591	-0.1493	.	0.0484	-0.7480	-0.0550	.	.	-0.0284	.

7	0.53101	5.293	0.38172	10.1563	0.1424	-0.1319	.	.	-0.7913	-0.0575	-0.0108	.	-0.0168	-0.0157
7	0.52851	5.555	0.38274	11.2157	0.1278	-0.1194	.	-0.0983	-0.7985	-0.0661	-0.0169	.	.	-0.0220
7	0.52769	5.641	0.38307	10.3654	0.1398	-0.1264	.	.	-0.8527	-0.0588	-0.0097	-0.0096	-0.0214	.
7	0.52669	5.746	0.38347	10.7349	0.1285	-0.1178	.	.	-0.8603	-0.0632	-0.0131	-0.00728	.	-0.0187
7	0.52452	5.975	0.38435	10.7156	0.1351	-0.1248	-0.00033	.	-0.8317	-0.0665	-0.0130	.	.	-0.0218
8	0.53269	7.116	0.38475	10.3449	0.1359	-0.1241	.	.	-0.8180	-0.0553	-0.0110	-0.00631	-0.0161	-0.0134
8	0.53188	7.201	0.38509	10.5849	0.1370	-0.1271	.	-0.0514	-0.7818	-0.0591	-0.0132	.	-0.0135	-0.0170
8	0.53179	7.211	0.38512	10.6858	0.1413	-0.1266	-0.00102	.	-0.7892	-0.0578	-0.0108	.	-0.0181	-0.0156
8	0.52978	7.422	0.38595	11.2915	0.1233	-0.1135	.	-0.0872	-0.8243	-0.0639	-0.0165	-0.00556	.	-0.0197
8	0.52870	7.536	0.38639	11.4933	0.1270	-0.1166	-0.00049	-0.0997	-0.7985	-0.0666	-0.0170	.	.	-0.0222
9	0.53332	9.050	0.38832	10.8118	0.1352	-0.1197	-0.00092	.	-0.8149	-0.0557	-0.0110	-0.00603	-0.0173	-0.0133
9	0.53320	9.063	0.38837	10.6575	0.1324	-0.1212	.	-0.0398	-0.8079	-0.0568	-0.0129	-0.00567	-0.0136	-0.0146
9	0.53260	9.125	0.38862	11.0776	0.1362	-0.1222	-0.00098	-0.0496	-0.7801	-0.0594	-0.0132	.	-0.0149	-0.0168
9	0.52991	9.409	0.38974	11.5195	0.1226	-0.1113	-0.0004	-0.0886	-0.8238	-0.0644	-0.0167	-0.00544	.	-0.0199
9	0.52835	9.572	0.39038	10.8569	0.1390	-0.1217	-0.00094	-0.00188	-0.8491	-0.0593	-0.0099	-0.00927	-0.0225	.
10	0.53379	11.000	0.39206	11.1043	0.1318	-0.1170	-0.00089	-0.0386	-0.8052	-0.0572	-0.0128	-0.00541	-0.0149	-0.0146

WWT = warm carcass weight; CWT = cold carcass weight; LNGTH = carcass length; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*

Annex B Table 6. Regression Models for prediction of Dependent Variable: Silverside as a percentage of cold carcass weight

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	LNGTH	PH45	PH24	FAT	MSCLE	T1	T2-3	GM
1	0.55525	18.188	0.37432	6.9451	.	.	.	.	.	-0.0989	.	.	.	.
1	0.47104	32.236	0.40822	7.1086	.	.	.	.	.	.	.	.	-0.0916	.
1	0.44736	36.185	0.41726	6.7737	.	.	.	.	.	.	.	.	.	-0.0783
1	0.33869	54.312	0.45644	7.3641	.	.	.	.	.	.	.	-0.0653	.	.
1	0.23354	71.852	0.49140	2.8024	.	.	.	.	.	.	0.0441	.	.	.
1	0.16987	82.473	0.51140	8.4988	.	.	.	-0.5316	.	.	.	.	.	.
1	0.14542	86.552	0.51888	12.1586	.	.	.	.	-1.2214	.	.	.	.	.
1	0.03916	104.3	0.55019	1.4379	.	.	0.00510	.	.	.	.	.	.	.
1	0.00059	110.7	0.56112	5.6011	.	-0.00366	.	.	.	.	.	.	.	.
1	0.00001	110.8	0.56128	5.4013	-0.00058	.	.	.	.	.	.	.	.	.
2	0.62983	7.748	0.34448	11.7796	.	.	.	.	-0.8846	-0.0934	.	.	.	.
2	0.60107	12.545	0.35761	7.2577	.	.	.	.	.	-0.0692	.	.	-0.0413	.
2	0.59028	14.345	0.36242	7.1002	.	.	.	.	.	-0.0728	.	.	.	-0.0318
2	0.58931	14.507	0.36284	8.2949	.	.	.	-0.2511	.	-0.0907	.	.	.	.
2	0.58020	16.026	0.36685	7.3725	.	.	.	.	.	-0.0826	.	-0.0224	.	.
3	0.66985	3.072	0.32822	12.8504	.	.	.	.	-0.9805	-0.0720	.	-0.0287	.	.
3	0.65948	4.801	0.33333	11.5356	.	.	.	.	-0.7933	-0.0698	.	.	-0.0337	.
3	0.64933	6.495	0.33826	11.4382	.	.	.	.	-0.8006	-0.0741	.	.	.	-0.0241
3	0.64253	7.629	0.34153	12.0574	.	.	.	-0.1597	-0.7783	-0.0889	.	.	.	.
3	0.63154	9.462	0.34674	12.1747	.	-0.00626	.	.	-0.8817	-0.0936	.	.	.	.
4	0.68785	2.069	0.32203	12.5063	.	.	.	.	-0.8944	-0.0562	.	-0.0247	-0.0268	.
4	0.67645	3.970	0.32786	12.9816	.	.	.	-0.1167	-0.8964	-0.0701	.	-0.0268	.	.
4	0.67529	4.164	0.32845	12.5087	.	.	.	.	-0.9197	-0.0641	.	-0.0247	.	-0.0136
4	0.67345	4.471	0.32938	11.6349	.	.	0.00162	.	-0.9863	-0.0688	.	-0.0303	.	.
4	0.67034	4.990	0.33094	12.6683	0.00343	.	.	.	-0.9876	-0.0715	.	-0.0292	.	.
5	0.69071	3.592	0.32351	12.6252	.	.	.	-0.0787	-0.8453	-0.0563	.	-0.0238	-0.0244	.
5	0.68983	3.740	0.32397	11.6134	.	.	0.00121	.	-0.9021	-0.0544	.	-0.0261	-0.0257	.
5	0.68921	3.842	0.32429	12.1914	0.00573	.	.	.	-0.9037	-0.0549	.	-0.0254	-0.0276	.
5	0.68890	3.894	0.32445	12.3760	.	.	.	.	-0.8733	-0.0538	.	-0.0232	-0.0246	-0.00631
5	0.68801	4.043	0.32492	12.3949	.	0.00195	.	.	-0.8959	-0.0557	.	-0.0251	-0.0270	.
6	0.70039	3.977	0.32140	12.4431	0.0784	-0.0743	.	.	-0.9644	-0.0568	.	-0.0204	-0.0293	.
6	0.69213	5.355	0.32580	12.4811	.	.	.	-0.0840	-0.8174	-0.0535	.	-0.0219	-0.0217	-0.00737
6	0.69207	5.366	0.32583	11.8655	.	.	0.00101	-0.0704	-0.8569	-0.0548	.	-0.0250	-0.0238	.
6	0.69135	5.486	0.32621	13.1483	.	.	.	-0.1030	-0.8732	-0.0598	-0.00364	-0.0235	-0.0225	.
6	0.69128	5.497	0.32624	12.4000	0.00384	.	.	-0.0693	-0.8574	-0.0554	.	-0.0243	-0.0253	.

7	0.70343	5.471	0.32282	11.3581	0.0799	-0.0846	0.00214	.	-0.9727	-0.0559	.	-0.0211	-0.0265	.
7	0.70196	5.715	0.32362	12.6198	0.0752	-0.0728	.	-0.0605	-0.9227	-0.0572	.	-0.0196	-0.0273	.
7	0.70092	5.889	0.32418	12.3882	0.0772	-0.0740	.	.	-0.9475	-0.0552	.	-0.0193	-0.0276	-0.00458
7	0.70067	5.931	0.32432	12.2068	0.0796	-0.0763	.	.	-0.9398	-0.0549	0.00211	-0.0202	-0.0300	.
7	0.69354	7.121	0.32816	13.2521	.	.	.	-0.1234	-0.8525	-0.0582	-0.00566	-0.0210	-0.0179	-0.0096
8	0.70511	7.189	0.32504	11.5208	0.0766	-0.0831	0.00218	-0.0626	-0.9296	-0.0563	.	-0.0203	-0.0243	.
8	0.70398	7.378	0.32567	11.2978	0.0787	-0.0842	0.00215	.	-0.9555	-0.0543	.	-0.0199	-0.0248	-0.00468
8	0.70372	7.421	0.32581	11.1110	0.0811	-0.0867	0.00215	.	-0.9473	-0.0540	0.00218	-0.0209	-0.0272	.
8	0.70284	7.568	0.32629	12.5693	0.0734	-0.0721	.	-0.0677	-0.8958	-0.0552	.	-0.0180	-0.0248	-0.00597
8	0.70199	7.710	0.32676	12.7258	0.0744	-0.0719	.	-0.0657	-0.9285	-0.0579	-0.00081	-0.0196	-0.0268	.
9	0.70604	9.036	0.32776	11.4611	0.0747	-0.0825	0.00220	-0.0700	-0.9021	-0.0542	.	-0.0186	-0.0217	-0.00612
9	0.70515	9.184	0.32826	11.6312	0.0758	-0.0822	0.00219	-0.0681	-0.9358	-0.0571	-0.00085	-0.0203	-0.0239	.
9	0.70417	9.346	0.32880	11.1012	0.0799	-0.0860	0.00216	.	-0.9363	-0.0529	0.00178	-0.0198	-0.0254	-0.00428
9	0.70304	9.535	0.32943	12.8507	0.0711	-0.0696	.	-0.0828	-0.9080	-0.0569	-0.00221	-0.0178	-0.0233	-0.00679
9	0.69993	10.054	0.33115	12.3125	0.0599	-0.0722	0.00287	-0.1535	-0.9399	-0.0658	-0.00749	-0.0185	.	-0.0142
10	0.70625	11.000	0.33097	11.7497	0.0724	-0.0799	0.00221	-0.0857	-0.9148	-0.0560	-0.00228	-0.0185	-0.0202	-0.00697

WWT = warm carcass weight; CWT = cold carcass weight; LNGTH = carcass length; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*

Annex B Table 7. Regression Models for prediction of Dependent Variable: Thickflank as a percentage of cold carcass weight

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	LNGTH	PH45	PH24	FAT	MSCLE	T1	T2-3	GM
1	0.35996	3.768	0.25072	3.6248	.	.	.	.	.	-0.0445	.	.	.	.
1	0.20694	18.057	0.27909	3.4492	.	.	.	.	.	.	.	.	.	-0.0297
1	0.18251	20.338	0.28336	3.5202	.	.	.	.	.	.	.	.	-0.0318	.
1	0.13750	24.541	0.29105	1.8165	.	.	.	.	.	.	0.0189	.	.	.
1	0.13058	25.187	0.29222	6.5092	.	.	.	.	-0.6463	.	.	.	.	.
1	0.11931	26.240	0.29411	3.5767	.	.	.	.	.	.	.	-0.0216	.	.
1	0.05427	32.314	0.30477	3.9031	.	.	.	-0.1678	.	.	.	.	.	.
1	0.04689	33.003	0.30596	0.5155	.	.	0.00311	.	.	.	.	.	.	.
1	0.00155	37.236	0.31315	3.1369	-0.00335	.	.	.	.	.	.	.	.	.
1	0.00053	37.332	0.31331	3.0401	.	-0.00195	.	.	.	.	.	.	.	.
2	0.43548	-1.285	0.23752	6.3413	.	.	.	.	-0.4971	-0.0414	.	.	.	.
2	0.36583	5.219	0.25175	2.7290	.	.	0.00113	.	.	-0.0431	.	.	.	.
2	0.36348	5.439	0.25222	3.9642	-0.00505	.	.	.	.	-0.0446	.	.	.	.
2	0.36192	5.585	0.25253	3.8056	.	.	.	-0.0336	.	-0.0434	.	.	.	.
2	0.36169	5.606	0.25257	3.8555	.	-0.00352	.	.	.	-0.0446	.	.	.	.
3	0.44146	0.157	0.23836	5.4394	.	.	0.00114	.	-0.4974	-0.0400	.	.	.	.
3	0.43972	0.320	0.23874	6.9048	.	.	.	.	-0.5441	-0.0446	-0.00431	.	.	.
3	0.43841	0.442	0.23901	6.3841	.	.	.	.	-0.5131	-0.0455	.	.	0.00590	.
3	0.43698	0.575	0.23932	6.5344	-0.00331	.	.	.	-0.4918	-0.0415	.	.	.	.
3	0.43676	0.596	0.23937	6.5315	.	-0.00302	.	.	-0.4957	-0.0415	.	.	.	.
4	0.45795	0.618	0.23695	4.9036	.	-0.0154	0.00306	.	-0.4909	-0.0381	.	.	.	.
4	0.45692	0.714	0.23717	4.9422	-0.0145	.	0.00285	.	-0.4748	-0.0385	.	.	.	.
4	0.44791	1.555	0.23913	5.9750	.	.	0.00136	.	-0.5565	-0.0438	-0.00541	.	.	.
4	0.44509	1.818	0.23974	5.4330	.	.	0.00121	.	-0.5153	-0.0445	.	.	0.00659	.
4	0.44437	1.885	0.23989	7.0784	.	.	.	.	-0.5746	-0.0506	-0.00522	.	0.00760	.
5	0.46727	1.747	0.23706	4.7855	.	-0.0185	0.00355	.	-0.5193	-0.0453	.	.	0.0109	.
5	0.46598	1.868	0.23735	4.8310	-0.0174	.	0.00330	.	-0.4994	-0.0456	.	.	0.0108	.
5	0.46040	2.389	0.23859	5.2989	.	-0.0138	0.00300	.	-0.5292	-0.0407	-0.00345	.	.	.
5	0.45990	2.435	0.23870	5.3638	-0.0130	.	0.00283	.	-0.5181	-0.0413	-0.00377	.	.	.
5	0.45894	2.525	0.23891	4.8222	.	-0.0150	0.00307	0.0253	-0.5079	-0.0388	.	.	.	.
6	0.47160	3.342	0.23831	5.3047	.	-0.0167	0.00354	.	-0.5740	-0.0496	-0.00464	.	0.0122	.
6	0.47111	3.388	0.23842	5.3782	-0.0159	.	0.00334	.	-0.5603	-0.0502	-0.00501	.	0.0122	.
6	0.46821	3.659	0.23908	4.8216	.	-0.0182	0.00362	.	-0.5299	-0.0438	.	-0.00258	0.0117	.
6	0.46787	3.691	0.23915	4.8657	-0.0175	.	0.00345	.	-0.5145	-0.0435	.	-0.00364	0.0119	.
6	0.46739	3.736	0.23926	4.7750	.	-0.0186	0.00356	.	-0.5166	-0.0447	.	.	0.0114	-0.00114

7	0.47333	5.181	0.24020	5.4336	-0.0160	.	0.00350	.	-0.5786	-0.0481	-0.00518	-0.00395	0.0134	.
7	0.47294	5.218	0.24029	5.6879	-0.0167	.	0.00342	-0.0421	-0.5568	-0.0518	-0.00683	.	0.0143	.
7	0.47285	5.226	0.24031	5.3650	.	-0.0164	0.00361	.	-0.5882	-0.0480	-0.00481	-0.00296	0.0130	.
7	0.47273	5.237	0.24034	5.5579	.	-0.0171	0.00357	-0.0329	-0.5721	-0.0508	-0.00608	.	0.0138	.
7	0.47205	5.301	0.24049	5.3098	.	-0.0169	0.00355	.	-0.5714	-0.0487	-0.00487	.	0.0132	-0.00224
8	0.47466	7.057	0.24224	5.6940	-0.0166	.	0.00355	-0.0362	-0.5735	-0.0497	-0.00672	-0.00351	0.0152	.
8	0.47432	7.088	0.24232	5.7459	-0.0174	.	0.00349	-0.0499	-0.5507	-0.0505	-0.00757	.	0.0167	-0.00404
8	0.47367	7.149	0.24247	5.5760	.	-0.0167	0.00363	-0.0284	-0.5848	-0.0493	-0.00603	-0.00260	0.0143	.
8	0.47355	7.161	0.24249	5.3922	-0.0105	-0.00600	0.00358	.	-0.5814	-0.0480	-0.00501	-0.00359	0.0134	.
8	0.47353	7.162	0.24250	5.6064	.	-0.0174	0.00360	-0.0383	-0.5683	-0.0498	-0.00663	.	0.0155	-0.00304
9	0.47522	9.004	0.24452	5.7324	-0.0172	.	0.00357	-0.0429	-0.5657	-0.0493	-0.00725	-0.00273	0.0166	-0.00277
9	0.47474	9.050	0.24463	5.6581	-0.0132	-0.00373	0.00360	-0.0348	-0.5755	-0.0496	-0.00656	-0.00331	0.0150	.
9	0.47449	9.072	0.24469	5.6912	-0.0124	-0.00535	0.00357	-0.0468	-0.5553	-0.0503	-0.00727	.	0.0164	-0.00379
9	0.47400	9.119	0.24480	5.6054	.	-0.0170	0.00363	-0.0332	-0.5792	-0.0489	-0.00642	-0.00198	0.0154	-0.00211
9	0.47370	9.147	0.24487	5.3890	-0.0109	-0.00578	0.00359	.	-0.5778	-0.0477	-0.00514	-0.00325	0.0139	-0.00139
10	0.47527	11.000	0.24699	5.7040	-0.0145	-0.00284	0.00361	-0.0416	-0.5673	-0.0492	-0.00711	-0.00259	0.0165	-0.00270

WWT = warm carcass weight; CWT = cold carcass weight; LNGTH = carcass length; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*

Annex B Table 8. Regression Models for prediction of Dependent Variable: Rump as a percentage of cold carcass weight

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	LNGTH	PH45	PH24	FAT	MSCLE	T1	T2-3	GM
1	0.20489	17.511	0.29856	1.6730	.	.	.	.	.	-0.0358	.	.	.	.
1	0.18172	19.653	0.30288	-0.2476	.	.	.	.	.	.	0.0232	.	.	.
1	0.16211	21.466	0.30649	1.9255	.	.	.	.	.	.	.	-0.0270	.	.
1	0.13577	23.901	0.31127	1.5632	.	.	.	.	.	.	.	.	.	-0.0257
1	0.07701	29.334	0.32167	2.3592	.	.	.	-0.2135	.	.	.	.	.	.
1	0.05384	31.476	0.32569	3.5663	.	.	.	.	-0.4434	.	.	.	.	.
1	0.04270	32.505	0.32760	1.4131	.	.	.	.	.	.	.	.	-0.0164	.
1	0.02533	34.111	0.33056	2.0346	.	-0.0143	.	.	.	.	.	.	.	.
1	0.00786	35.727	0.33351	1.6368	-0.00806	.	.	.	.	.	.	.	.	.
1	0.00768	35.744	0.33354	2.1363	.	.	-0.00135	.	.	.	.	.	.	.
2	0.26665	13.800	0.28923	0.6565	.	.	.	.	.	.	0.0184	-0.0204	.	.
2	0.25517	14.863	0.29149	1.1790	.	-0.0252	.	.	.	.	0.0269	.	.	.
2	0.24827	15.500	0.29284	0.6993	.	.	.	.	.	-0.0247	0.0137	.	.	.
2	0.24509	15.794	0.29345	4.1766	.	.	-0.00317	.	.	-0.0397	.	.	.	.
2	0.23518	16.710	0.29537	1.5213	.	.	.	.	.	-0.0502	.	.	0.0200	.
3	0.37521	5.764	0.26934	0.5822	0.1371	-0.1584	.	.	.	.	0.0293	.	.	.
3	0.31321	11.496	0.28239	2.2747	0.1099	-0.1216	.	.	.	-0.0360	.	.	.	.
3	0.31054	11.743	0.28294	1.6360	.	-0.0200	.	.	.	.	0.0221	-0.0169	.	.
3	0.30549	12.210	0.28397	1.8532	.	-0.0224	.	.	.	-0.0216	0.0181	.	.	.
3	0.30514	12.242	0.28404	3.5309	.	.	-0.00382	.	.	-0.0272	0.0163	.	.	.
4	0.41141	4.417	0.26379	1.1903	0.1294	-0.1486	.	.	.	-0.0184	0.0217	.	.	.
4	0.39533	5.904	0.26737	0.9430	0.1203	-0.1388	.	.	.	.	0.0260	-0.0106	.	.
4	0.38911	6.479	0.26874	0.9560	0.1306	-0.1513	.	.	.	.	0.0254	.	.	-0.0096
4	0.38746	6.632	0.26910	1.9327	0.1460	-0.1657	.	.	-0.2359	.	0.0266	.	.	.
4	0.37588	7.702	0.27163	0.6315	0.1371	-0.1580	.	.	.	.	0.0287	.	-0.00222	.
5	0.43161	4.550	0.26161	1.2450	0.1236	-0.1442	.	.	.	-0.0313	0.0206	.	0.0167	.
5	0.42782	4.900	0.26248	2.7965	0.1393	-0.1565	.	.	-0.2742	-0.0196	0.0181	.	.	.
5	0.41674	5.925	0.26501	2.0736	0.1270	-0.1395	-0.00164	.	.	-0.0205	0.0214	.	.	.
5	0.41330	6.243	0.26579	1.2304	0.1244	-0.1429	.	.	.	-0.0156	0.0216	-0.00392	.	.
5	0.41267	6.301	0.26593	2.6132	0.1288	-0.1451	.	.	-0.2835	.	0.0224	-0.0120	.	.
6	0.45701	4.201	0.25810	3.2961	0.1348	-0.1533	.	.	-0.3480	-0.0356	0.0159	.	0.0204	.
6	0.43674	6.075	0.26287	1.3178	0.1146	-0.1342	.	.	.	-0.0278	0.0204	-0.00659	0.0183	.
6	0.43634	6.112	0.26297	1.3741	0.1193	-0.1405	.	.	.	-0.0285	0.0196	.	0.0205	-0.00785
6	0.43559	6.182	0.26314	5.4760	0.1184	-0.1348	.	-0.1368	-0.4387	-0.0504	.	.	0.0300	.
6	0.43475	6.260	0.26334	1.7843	0.1185	-0.1397	.	-0.0599	.	-0.0339	0.0178	.	0.0198	.

7	0.46641	5.332	0.25830	3.5871	0.1235	-0.1404	.	.	-0.3805	-0.0313	0.0152	-0.00902	0.0230	.
7	0.46001	5.924	0.25985	3.3360	0.1311	-0.1501	.	.	-0.3373	-0.0333	0.0152	.	0.0234	-0.00628
7	0.45905	6.013	0.26008	3.6908	0.1305	-0.1495	.	-0.0483	-0.3412	-0.0376	0.0137	.	0.0229	.
7	0.45842	6.071	0.26023	3.7478	0.1339	-0.1488	-0.00087	.	-0.3460	-0.0359	0.0159	.	0.0194	.
7	0.44688	7.138	0.26299	5.4387	0.1110	-0.1291	.	-0.1471	-0.3961	-0.0446	.	.	0.0357	-0.0121
8	0.46771	7.213	0.26051	3.8927	0.1205	-0.1378	.	-0.0387	-0.3738	-0.0330	0.0135	-0.00869	0.0248	.
8	0.46731	7.249	0.26060	3.9418	0.1230	-0.1371	-0.0007	.	-0.3780	-0.0316	0.0152	-0.00880	0.0221	.
8	0.46713	7.265	0.26065	3.5822	0.1226	-0.1399	.	.	-0.3721	-0.0304	0.0149	-0.00823	0.0243	-0.00322
8	0.46360	7.592	0.26151	3.8864	0.1242	-0.1439	.	-0.0660	-0.3250	-0.0354	0.0120	.	0.0275	-0.00796
8	0.46130	7.805	0.26207	3.7667	0.1302	-0.1458	-0.00083	.	-0.3355	-0.0336	0.0152	.	0.0223	-0.00615
9	0.46919	9.075	0.26273	3.9814	0.1181	-0.1362	.	-0.0508	-0.3593	-0.0323	0.0125	-0.00741	0.0274	-0.00482
9	0.46856	9.134	0.26289	4.2317	0.1201	-0.1346	-0.00068	-0.0379	-0.3716	-0.0333	0.0135	-0.00849	0.0239	.
9	0.46802	9.183	0.26302	3.9344	0.1221	-0.1366	-0.00069	.	-0.3698	-0.0308	0.0149	-0.00802	0.0234	-0.00319
9	0.46472	9.488	0.26384	4.2770	0.1235	-0.1400	-0.00078	-0.0646	-0.3237	-0.0356	0.0121	.	0.0264	-0.00780
9	0.45198	10.667	0.26696	5.8963	0.1048	-0.1188	-0.00064	-0.1358	-0.4268	-0.0425	.	-0.00634	0.0349	-0.00940
10	0.47000	11.000	0.26520	4.3127	0.1177	-0.1331	-0.00066	-0.0500	-0.3572	-0.0326	0.0125	-0.00722	0.0264	-0.00477

WWT = warm carcass weight; CWT = cold carcass weight; LNGTH = carcass length; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*

Annex B Table 9. Regression Models for prediction of Dependent Variable: Shoulder fat/skin as a percentage of cold carcass weight

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	LNGTH	PH45	PH24	FAT	MSCLE	T1	T2-3	GM
1	0.40427	16.404	0.56009	1.9589	.	.	.	.	.	.	.	.	.	0.0962
1	0.38652	18.561	0.56837	0.9288	.	.	.	.	.	.	.	0.0902	.	.
1	0.32716	25.776	0.59523	2.1227	.	.	.	.	.	0.0982	.	.	.	.
1	0.24872	35.310	0.62897	7.1089	.	.	.	.	.	.	-0.0588	.	.	.
1	0.22376	38.343	0.63933	2.1373	.	.	.	.	.	.	.	.	0.0816	.
1	0.18879	42.594	0.65358	-0.5814	.	.	.	0.7245	.	.	.	.	.	.
1	0.07953	55.872	0.69620	-2.8047	.	.	.	.	1.1678	.	.	.	.	.
1	0.00199	65.297	0.72493	4.2791	-0.00879	.	.	.	.	.	.	.	.	.
1	0.00113	65.402	0.72525	4.5536	.	.	-0.00112	.	.	.	.	.	.	.
1	0.00058	65.467	0.72544	4.0011	.	-0.00472	.	.	.	.	.	.	.	.
2	0.49712	7.119	0.51908	3.7576	.	.	.	.	.	.	-0.0410	0.0756	.	.
2	0.48927	8.074	0.52312	0.9351	.	.	.	.	.	.	.	0.0537	.	0.0616
2	0.47420	9.905	0.53078	-5.9252	.	.	.	.	1.2265	.	.	0.0912	.	.
2	0.47287	10.067	0.53146	-0.5169	.	.	.	0.4562	.	.	.	.	.	0.0843
2	0.45998	11.633	0.53791	-1.4971	.	.	.	0.4708	.	.	.	0.0787	.	.
3	0.53965	3.950	0.50106	-1.0987	.	.	.	0.3946	.	.	.	0.0481	.	0.0549
3	0.53716	4.253	0.50242	2.9811	.	.	.	.	.	.	-0.0297	0.0546	.	0.0423
3	0.53172	4.914	0.50536	-4.0883	.	.	.	.	0.8987	.	.	0.0622	.	0.0486
3	0.52763	5.412	0.50757	-1.3500	.	.	.	.	0.7939	.	-0.0312	0.0797	.	.
3	0.50805	7.790	0.51798	-5.0293	.	.	.	.	1.0654	0.0409	.	0.0698	.	.
4	0.56082	3.377	0.49384	-4.4029	.	.	.	0.3143	0.6651	.	.	0.0555	.	0.0466
4	0.55748	3.784	0.49571	-1.1534	.	.	.	.	0.6575	.	-0.0230	0.0606	.	0.0371
4	0.55288	4.342	0.49828	0.8626	.	.	.	0.2630	.	.	-0.0186	0.0505	.	0.0451
4	0.55036	4.649	0.49968	-4.0671	.	.	0.00364	0.4256	.	.	.	0.0443	.	0.0603
4	0.54837	4.891	0.50079	0.2838	.	.	0.00374	.	.	.	-0.0323	0.0512	.	0.0467
5	0.56986	4.278	0.49323	-4.6214	.	.	.	0.3621	0.6832	.	.	0.0584	-0.0246	0.0586
5	0.56844	4.451	0.49405	-2.4716	.	.	.	0.2223	0.5824	.	-0.0144	0.0564	.	0.0400
5	0.56838	4.458	0.49408	-6.6803	.	.	0.00308	0.3463	0.6180	.	.	0.0517	.	0.0518
5	0.56651	4.685	0.49515	-2.2877	.	.	0.00413	0.2835	.	.	-0.0207	0.0464	.	0.0501
5	0.56526	4.837	0.49586	-3.0882	.	.	0.00314	.	0.6047	.	-0.0258	0.0572	.	0.0412
6	0.58384	4.579	0.48971	-7.7922	.	-0.0349	0.00711	0.3165	0.6761	.	.	0.0566	.	0.0513
6	0.58242	4.752	0.49054	-7.6393	-0.0321	.	0.00658	0.3141	0.7080	.	.	0.0549	.	0.0515
6	0.57905	5.161	0.49252	-4.4060	-0.0319	.	0.00659	.	0.6993	.	-0.0230	0.0599	.	0.0420
6	0.57841	5.239	0.49289	-4.8063	.	.	0.00357	0.2448	0.5146	.	-0.0167	0.0522	.	0.0449
6	0.57811	5.275	0.49307	-4.5263	.	-0.0323	0.00678	.	0.6747	.	-0.0223	0.0615	.	0.0421

7	0.59038	5.784	0.49049	-6.1378	.	-0.0311	0.00707	0.2366	0.5852	.	-0.0137	0.0565	.	0.0457
7	0.59024	5.802	0.49058	-5.9019	-0.0296	.	0.00675	0.2265	0.6093	.	-0.0148	0.0551	.	0.0454
7	0.58928	5.919	0.49115	-8.0744	.	-0.0371	0.00776	0.3016	0.6728	0.0198	.	0.0522	.	0.0425
7	0.58836	6.030	0.49170	-7.9476	-0.0349	.	0.00732	0.2975	0.7086	0.0208	.	0.0502	.	0.0423
7	0.58633	6.277	0.49291	-7.6382	.	-0.0301	0.00638	0.3455	0.6806	.	.	0.0578	-0.0137	0.0577
8	0.59604	7.096	0.49184	-7.9245	.	-0.0294	0.00677	0.3465	0.6793	0.0284	.	0.0523	-0.0243	0.0501
8	0.59488	7.237	0.49255	-7.8003	-0.0270	.	0.00634	0.3437	0.7055	0.0290	.	0.0507	-0.0241	0.0499
8	0.59262	7.513	0.49392	-6.7154	.	-0.0335	0.00753	0.2448	0.6040	0.0137	-0.0105	0.0535	.	0.0409
8	0.59256	7.519	0.49396	-6.4785	-0.0321	.	0.00721	0.2340	0.6307	0.0139	-0.0117	0.0519	.	0.0405
8	0.59155	7.642	0.49457	-6.1754	.	-0.0281	0.00657	0.2639	0.5964	.	-0.0125	0.0573	-0.0096	0.0507
9	0.59683	9.001	0.49626	-7.2318	.	-0.0286	0.00679	0.3102	0.6422	0.0239	-0.00552	0.0530	-0.0208	0.0482
9	0.59613	9.086	0.49668	-6.9546	-0.0268	.	0.00646	0.2971	0.6598	0.0233	-0.00694	0.0516	-0.0195	0.0474
9	0.59605	9.095	0.49673	-7.9269	0.00284	-0.0322	0.00678	0.3473	0.6761	0.0283	.	0.0524	-0.0244	0.0502
9	0.59291	9.477	0.49866	-6.6378	-0.0157	-0.0179	0.00747	0.2394	0.6190	0.0139	-0.0110	0.0528	.	0.0407
9	0.59160	9.636	0.49946	-6.1353	-0.00675	-0.0215	0.00656	0.2603	0.6021	.	-0.0128	0.0570	-0.00915	0.0504
10	0.59684	11.000	0.50129	-7.2114	-0.00300	-0.0257	0.00679	0.3085	0.6446	0.0239	-0.00566	0.0528	-0.0206	0.0481

WWT = warm carcass weight; CWT = cold carcass weight; LNGTH = carcass length; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*

Annex B Table 10. Regression Models for prediction of Dependent Variable: Shoulder lean as a percentage of cold carcass weight

In	Rsq	C(p)	Root MSE	Parameter Estimates Intercept	WWT	CWT	LNGTH	PH45	PH24	FAT	MSCLE	T1	T2-3	GM
1	0.18045	-4.598	0.76908	17.2622	.	.	.	.	.	-0.0853	.	.	.	.
1	0.12261	-0.971	0.79575	17.0148	.	.	.	.	.	.	.	.	.	-0.0620
1	0.07273	2.158	0.81806	17.3005	.	.	.	.	.	.	.	-0.0458	.	.
1	0.06662	2.541	0.82075	16.8905	.	.	.	.	.	.	.	.	-0.0521	.
1	0.05506	3.266	0.82582	14.0152	.	.	.	.	.	.	0.0324	.	.	.
1	0.01205	5.963	0.84440	17.5331	.	-0.0251	.	.	.	.	.	.	.	.
1	0.01150	5.998	0.84464	17.5460	-0.0247	.	.	.	.	.	.	.	.	.
1	0.00546	6.377	0.84722	16.7475	.	.	.	-0.1442	.	.	.	.	.	.
1	0.00497	6.407	0.84742	17.7989	.	.	.	.	-0.3419	.	.	.	.	.
1	0.00036	6.696	0.84938	16.4683	.	.	-0.00074	.	.	.	.	.	.	.
2	0.19561	-3.549	0.76859	21.1630	.	.	-0.00494	.	.	-0.0914	.	.	.	.
2	0.19557	-3.547	0.76860	19.1087	.	-0.0281	.	.	.	-0.0861	.	.	.	.
2	0.19519	-3.523	0.76879	19.1436	-0.0280	.	.	.	.	-0.0862	.	.	.	.
2	0.18542	-2.910	0.77344	17.1063	.	.	.	.	.	-0.1002	.	.	0.0206	.
2	0.18463	-2.861	0.77381	16.5458	.	.	.	0.1333	.	-0.0897	.	.	.	.
3	0.20315	-2.022	0.77177	19.0840	.	-0.0307	.	.	.	-0.1046	.	.	0.0256	.
3	0.20313	-2.021	0.77178	19.1469	-0.0310	.	.	.	.	-0.1051	.	.	0.0263	.
3	0.20077	-1.873	0.77292	21.4411	.	.	-0.00518	.	.	-0.0764	.	.	.	-0.0185
3	0.20037	-1.848	0.77312	19.2603	.	-0.0291	.	.	.	-0.0715	.	.	.	-0.0178
3	0.20024	-1.840	0.77318	19.3181	-0.0293	.	.	.	.	-0.0711	.	.	.	-0.0183
4	0.21674	-0.875	0.77209	19.4616	-0.0350	.	.	.	.	-0.0891	.	.	0.0414	-0.0328
4	0.21595	-0.825	0.77248	19.3398	.	-0.0339	.	.	.	-0.0890	.	.	0.0400	-0.0317
4	0.21000	-0.452	0.77540	21.1147	.	.	-0.00499	.	.	-0.0897	.	.	0.0304	-0.0287
4	0.20438	-0.099	0.77816	20.1941	-0.0230	.	-0.00198	.	.	-0.1055	.	.	0.0237	.
4	0.20412	-0.0831	0.77828	20.0418	.	-0.0231	-0.00182	.	.	-0.1050	.	.	0.0234	.
5	0.21753	1.076	0.77881	20.2933	-0.0286	.	-0.00158	.	.	-0.0896	.	.	0.0391	-0.0323
5	0.21745	1.081	0.77885	19.4272	-0.0361	.	.	.	.	-0.0915	.	0.00612	0.0411	-0.0352
5	0.21732	1.089	0.77892	19.3277	.	-0.0360	.	.	.	-0.0924	.	0.00863	0.0398	-0.0351
5	0.21690	1.116	0.77913	19.2766	-0.0344	.	.	0.0276	.	-0.0892	.	.	0.0401	-0.0327
5	0.21678	1.123	0.77919	19.4585	-0.0290	-0.00611	.	.	.	-0.0891	.	.	0.0412	-0.0327
6	0.21851	3.014	0.78563	20.3948	-0.0287	.	-0.00186	.	.	-0.0926	.	0.00728	0.0384	-0.0350
6	0.21816	3.036	0.78581	20.2213	.	-0.0289	-0.00171	.	.	-0.0931	.	0.00917	0.0375	-0.0349
6	0.21773	3.063	0.78602	20.1079	-0.0276	.	-0.00163	0.0311	.	-0.0898	.	.	0.0376	-0.0321
6	0.21769	3.066	0.78605	19.4124	-0.0202	-0.0163	.	.	.	-0.0920	.	0.00733	0.0406	-0.0352
6	0.21758	3.073	0.78610	19.1123	-0.0364	.	.	.	0.0604	-0.0915	.	0.00679	0.0408	-0.0359

7	0.21864	5.006	0.79308	20.0818	-0.0290	.	-0.00185	.	0.0598	-0.0926	.	0.00794	0.0381	-0.0357
7	0.21863	5.007	0.79309	20.2491	-0.0280	.	-0.00188	0.0238	.	-0.0926	.	0.00700	0.0373	-0.0347
7	0.21857	5.011	0.79312	20.4576	-0.0284	.	-0.00184	.	.	-0.0937	-0.00136	0.00745	0.0389	-0.0355
7	0.21857	5.011	0.79312	20.3291	-0.0212	-0.00816	-0.00174	.	.	-0.0927	.	0.00781	0.0383	-0.0351
7	0.21830	5.028	0.79326	20.0654	.	-0.0282	-0.00174	0.0262	.	-0.0931	.	0.00881	0.0363	-0.0346
8	0.21870	7.003	0.80079	20.0427	-0.0284	.	-0.00187	0.0174	0.0469	-0.0926	.	0.00759	0.0373	-0.0354
8	0.21868	7.004	0.80080	20.1847	-0.0205	-0.00808	-0.00177	0.0237	.	-0.0927	.	0.00753	0.0372	-0.0348
8	0.21867	7.004	0.80081	20.0591	-0.0233	-0.00616	-0.00177	.	0.0547	-0.0927	.	0.00829	0.0380	-0.0357
8	0.21865	7.005	0.80082	20.1526	-0.0288	.	-0.00185	.	0.0521	-0.0931	-0.00066	0.00794	0.0383	-0.0359
8	0.21863	7.007	0.80083	20.2803	-0.0280	.	-0.00187	0.0212	.	-0.0928	-0.00034	0.00707	0.0375	-0.0349
9	0.21873	9.000	0.80874	20.0168	-0.0223	-0.00660	-0.00178	0.0182	0.0408	-0.0927	.	0.00795	0.0373	-0.0353
9	0.21870	9.003	0.80876	20.0201	-0.0284	.	-0.00187	0.0187	0.0481	-0.0924	0.000185	0.00757	0.0372	-0.0353
9	0.21868	9.004	0.80877	20.1717	-0.0204	-0.00824	-0.00177	0.0246	.	-0.0926	0.000126	0.00752	0.0371	-0.0347
9	0.21868	9.004	0.80877	20.1137	-0.0236	-0.00568	-0.00177	.	0.0493	-0.0931	-0.00049	0.00826	0.0382	-0.0358
9	0.21835	9.024	0.80894	19.8004	.	-0.0285	-0.00175	0.0345	0.0260	-0.0919	0.00157	0.00882	0.0353	-0.0343
10	0.21874	11.000	0.81695	19.9480	-0.0217	-0.00722	-0.00178	0.0219	0.0438	-0.0923	0.000544	0.00791	0.0369	-0.0351

WWT = warm carcass weight; CWT = cold carcass weight; LNGTH = carcass length; pH45 = initial pH; pH24 = ultimate pH; FAT = fat thickness between 2-3 last thoracic vertebrae 45 mm from midline; MSCLE = MLT depth between 2-3 last thoracic vertebrae 45 mm from midline; T1 = fat thickness at 1st thoracic vertebrae; T2-3 = fat thickness between 2-3 last thoracic vertebrae; GM = fat thickness over *M. gluteus medius*

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